Is chronic pain a small-t trauma?

A systematic review of the use of EMDR in the treatment of chronic pain.

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Dissertation submitted to the University of Chester for the Degree of Master of Arts (Clinical Counselling) in part fulfillment of the Modular Programme in Counselling. October 2012.

ABSTRACT

***Aim*:** Chronic pain (CP) hugely impacts negatively on the individual. Similarities between post-traumatic stress disorder (PTSD) and CP include neuro-plasticity, affect and memory, suggesting CP is a small-t trauma with PTSD a big-T Trauma. As such there is a theoretical rationale for the use of eye movement desensitisation and reprocessing (EMDR) in CP treatment.

Methodology: A systematic review of the available literature (eight papers) identified two different EMDR protocols. Standard EMDR protocol was used in phantom limb pain (PLP) subjects. Pain protocol EMDR was used in headache, fibromyalgia, and musculoskeletal pain subjects. The papers varied greatly in robustness.

Results: PLP subjects had higher pain intensity scores pre-intervention and lower pain intensity scores post intervention compared to other CP subjects. Both EMDR protocols demonstrated significant pain reduction/amelioration, maintained at follow-up. Further research is required; however this systematic review offers that EMDR has the potential to be a useful adjunct in CP management and treatment.

DECLARATION

This work is original and has not been submitted previously in support of any other qualification or course.

Signed……...Kim Louise Patel

ACKNOWLEDGEMENTS

Thanks to Himanshu and Rohan for giving me the space, time and computer to study and to Hugo for the daily walks and agility.

Thanks to my “doggy” friends for still being there after my self-imposed exile.

To Hazel, who inspired and encouraged me to move- a beacon of light in those dark, lonely, terrifying days. I am forever indebted.

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List of abbreviations

Abbreviation	Meaning
AIP	Adaptive Information Processing
BACP	British Association for Counselling and Psychotherapy
CBT	Cognitive Behavioural Therapy
CNS	Central Nervous System
CP	Chronic Pain
CRD	Centre for Reviews and Dissemination
DSM-IV (-TR)	Diagnostic and Statistical Manual of Mental Disorders
EBP	Evidence Based Practice
EMDR	Eye Movement Desensitization and Reprocessing
HPA-axis	Hypothalamic Pituitary Adrenal axis
IASP	International Association for the Study of Pain
ICD-10	International Classification of Diseases and Related Health
LAPS	Limbically Augmented Pain Syndrome
NICE	National Institute for Health and Clinical Excellence
NHS	National Health Service

PCC	Person-Centred Counselling/Counsellor
PLP	Phantom Limb Pain
PNS	Parasympathetic Nervous System
PP	Pain Protocol (EMDR)
PTSD	Post-Traumatic Stress Disorder
RCT	Randomized Controlled Trial
SFMQ	Short-Form McGill Pain Questionnaire
SNS	Sympathetic Nervous System
SP	Standard Protocol (EMDR)
SR	Systematic Review
SUD	Subjective Units of Distress
UK	United Kingdom

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INTRODUCTION

I have worked as a Clinical Nurse Specialist in Pain Management providing evidence based care to those suffering acute and/or chronic pain. Ironically, whilst in this role my own chronic pain (CP) developed and has been a part of my self for five years, at time of writing.

My early experiencing of CP was Descartian. I later learned and appreciated the interconnectedness of mind and body; thus for me, the self is embodied. I have experienced myriad emotions and learnt an inordinate amount about myself throughout this pain journey including strategies and techniques to improve physical and cognitive functioning, alongside person-centred counselling (PCC) and a cognitive-behavioural approach to movement, delivered through a pain management programme.

Whilst on professional placement as a trainee PCC, I worked with a gentleman (pseudonym Nye), who was struggling to function with post-traumatic stress disorder (PTSD). In our work I noticed parallels between my early pain journey and Nye's experiencing. Similarities were in our emotions, and physical and cognitive coping strategies. Cognitively, emotionally and behaviourally our recoveries, despite the differing causes, appeared and felt almost identical, leading me to question whether physiologically and psychologically there are similarities between PTSD and CP.

Furthermore, Nye wanted to work in a person-centred way and was also seeking (and setting himself) behavioural and cognitive challenges within and between our sessions. I felt a tension between working in a person-centred way and the cognitive/behavioural way that Nye was seeking. Support from my Supervisor and university tutor, specialising in PTSD counselling, enabled me to work effectively with Nye. This experience acted as a catalyst to exploring my personal and professional beliefs around uni-modal counselling; and the catalyst for this dissertation.

I intend to explore the similarities between CP and PTSD and the application of eye movement desensitization and reprocessing (EMDR) (a National Institute for Health and Clinical Excellence (NICE, 2005) recommended treatment for PTSD) in CP. The research question being:

Can CP be effectively treated using EMDR?

This is addressed through a review (a critical amalgamation of the summarised data) and synthesis (amalgamation of the concepts into a theoretical whole) of the literature (Dixon-woods, Argarwal, Jones, Young, Sutton, 2005; Noblit & Hare 1988; Petticrew & Roberts, 2006). Breaking tradition, the first two chapters offer an introduction to the relevant background areas and concepts pertinent to the systematic review.

In Chapter 1 I shall examine co-occurrence of PTSD and CP and the potential mechanisms, discussing brain plasticity, emotions and memory. Chapter 2 addresses the rationale for EMDR in relation to CP; Chapter 3 discusses the

research methodology, rationale, and ethical concerns. Chapter 4 presents the findings; with discussion, conclusion and areas for future research in Chapter 5. Appendices contain supportive information. This dissertation is concerned with similarities and areas of overlap between PTSD and CP, thus where the two conditions may differ and diverge is not addressed here.

The burden of CP on individuals and society is huge. Back pain accounts for 20% of the UK's total health expenditure. The prevalence is estimated at 8-60% of the population, depending on the definition (National Pain Audit, 2011). Severe pain is estimated at 11% for adults, and 8% for children. The average annual incidence is 8.3% and average annual recovery rate 5.4%; highlighting 3.1% a year will develop intractable pain. CP is known to have adverse effects on employment status, daily activities, relationships, mood, sleep and all aspects of general health (National Pain Audit, 2011). Daily back pain is known to be associated with greater coronary events. Provision of UK wide multidisciplinary pain management services are inconsistent - often too late and under resourced (National Pain Audit, 2011).

Individuals, in my experience, seek amelioration from CP outside of the NHS through for example, osteopaths and acupuncture. Suggesting medication alone is insufficient to manage pain, highlighting lack of specialist multidisciplinary services and implying lack of social/emotional support. The application of EMDR in CP treatment within and outside (independent) of the NHS provides alternative treatment of this complex phenomenon, allowing individuals greater choice in self-management.

It is also hoped that this dissertation, and any published articles from it, will provide information about the experiencing of CP and PTSD that may inform practice regardless of the modality practiced by counsellors. By increasing awareness of CP and PTSD mechanisms either as independent conditions or co-occurring I hope these conditions may be higher in practitioner's consciousness leading to improved management, and improved social and occupational functioning with less suffering for individuals.

CHAPTER ONE.

CHRONIC PAIN AND POST TRAUMATIC STRESS DISORDER: CO-OCCURRENCE AND AREAS OF SIMILARITIES.

Posttraumatic stress disorder, overview

The National Institute for Health and Clinical Excellence (NICE, 2005) describe PTSD developing "following a stressful event or situation of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone" (p.6). What is "exceptionally threatening or catastrophic" to an individual is subjective. The International Classification of Diseases and Related Health (ICD-10) (ICD-10, 1992, section F43.1) supplements 'a delayed response' to the NICE (2005) definition of PTSD, with a latency period from a few weeks to a few months and the duration of PTSD may be chronic or acute.

The ICD-10 (1992) describes PTSD as having predisposing factors such as personality traits or a previous history of neurotic illness. Features of PTSD include episodes of repeated reliving of the trauma via intrusive memories (flashbacks), dreams or nightmares, emotional numbness, detachment from others, unresponsiveness to surroundings, anhedonia and avoidance of trauma related stimuli. PTSD consists of a state of autonomic hyperarousal with hypervigilance, an enhanced startle reaction and insomnia. Anxiety and depression can occur alongside suicidal ideation (ICD-10, 1992).

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (APA, 2000) lists similar diagnostic features of PTSD, including social, occupational, and functional impairment.

Traumatic events injure a person's dignity and sense of integrity, leading to a sense of being damaged and/or incomplete. PTSD also challenges a person's beliefs about the world, their worthiness and significance (Herman, 1992). It leads to a process of withdrawal and disconnection from the person's world, their self, and meaningful connections to family, and social and spiritual interactions (Herman, 1992). Implicit is living with fear.

Chronic pain, overview

Melzack and Wall's (1965) Gate Control Theory of Pain, for the first time, acknowledged the importance of physiological, social, behavioural, and psychological influences, demonstrating the role of emotions in pain amplification and meaning (Gatchel, Peng, Peters, Fuchs, Turk, 2007). Expanded in Melzack's Neuromatrix Theory (1996) highlighting the role of memory and neural networks in the brain which maintain pain. Furthermore, Rome and Rome's (2000) Limbically Augmented Pain Syndrome (LAPS) emphasises the role of the limbic system (stress and pain experiences) in sensitizing the nervous system to pain.

The International Association for the Study of Pain (IASP, 1994) defines pain as "an unpleasant sensory or emotional experience associated with actual or potential tissue damage or described in terms of such damage" (www.iasp-pain.org), acknowledging that tissue damage is neither necessary nor sufficient for pain to exist (Butler & Moseley, 2003).

Acute pain is adaptive, alerting us to potential or actual tissue damage, motivating action to limit further injury and begin a process of recovery (Wall, 1978). As healing

occurs pain abates. For some, the pain does not subside, despite injury resolution, and is termed chronic if persisting for three months or more (IASP, 1986). CP has no adaptive qualities, causing considerable emotional distress and impairment of social and occupational functioning (Asmundson & Katz, 2009).

Co-occurrence & symptom overlap in PTSD and CP

Individuals with PTSD may present with several concomitant physical and mental health problems (Resnick, Yehuda, & Acierno, 1991; Schnurr & Jankowski, 1999). Recent evidence suggests pain is one of the commonly reported symptoms of individuals with PTSD regardless of the nature of their traumatic experience (e.g. combat, motor vehicle accident, sexual assault) (Asmundson, Coons, Taylor, Katz, 2002). Similarly individuals with persistent CP associated with a musculo-skeletal injury, serious burns and other pathologies (fibromyalgia, cancer, Acquired Human Immunity Deficiency Syndrome (AIDS)) frequently present with symptoms of PTSD (Asmundson et al. 2002).

Symptoms of PTSD and CP commonly co-occur (Blanchard, Hickling, Freidenberg, Malta, Kuhn, & Sykes, 2004; Coffey, Gudmundsdottir, Beck, Palyo & Miller, 2006; Geisser, Roth, Bachman & Eckert, 1996; Mayou, Ehlers & Bryant, 2002; McLean, Clauw, Abelson & Liberzon, 2005) creating vicious cycles of impairment and reduced quality of life (Miller, 2000). Models proposed to explain this co-occurrence include shared vulnerability (Asmundson et al. 2002; Otis, Keane, Kerns, 2003) and mutual maintenance (Sharp & Harvey, 2001) Appendix I.

Although the mechanisms for co-occurrence of PTSD and CP remain unclear, (Starr & Moulds, 2006) it is believed that anxiety sensitivity is implicated in each proposed model (Asmundson et al. 2002; Otis et al. 2003; Sharp & Harvey, 2001). The co-occurrence of PTSD and CP indicates emotional, behavioural, cognitive and physiological common ground in the two conditions.

Emotions are of adaptive value in dealing with fundamental life tasks (Ekman, 1999; Lazarus, 1991), unsurprisingly, emotion is considered one of the three components of the pain experience together with sensory and evaluative dimensions (Melzack & Katz, 2001), and is also inextricably linked with the PTSD experience (Mueller, 2007).

Power and Dalgleish (2008) propose that five "basic emotions" (happiness, sadness, anger, fear and disgust) are the building blocks of all emotional life (Ekman, 1992; Izard, 1991; Oatley & Johnson-Laird, 1987). The emotional response to pain (aside from its intrinsic unpleasantness) is anger, fear and sadness (Fernandez & Milburn, 1994). Anger expression and suppression are linked to pain severity via various physiological mechanisms (Bruehl, Chung, Burns, Biridepalli, 2003; Burns, Kubilus, Bruehl, 2003; Burns, Quartana, Bruehl, 2008). Fear of pain has also been closely related to various measures of patient functioning in CP (Crombez, Vlaeyen, Heuts, Lysens, 1999; McCracken, Zayfert, Cross, 1992) alongside anxiety sensitivity (Keogh & Cochrane, 2002) and worry (Eccleston & Crombez, 1999).

Several studies link sadness, anger and disgust with PTSD (Andrews, Brewin, Rose, Kirk, 2000; Budden, 2009; Dalgleish & Power, 2004; Olatunji, Ciessielski, Tolin, 2010; Van Vliet, 2008). In particular, expressing anger inwards and through verbal or

physical behaviour distinguishes those with PTSD from those with more general anxiety disorders (Finucane, Dima, Ferreira, Halvorsen, 2012).

Fear of pain can be more disabling than the pain itself (Crombez et al. 1999). The fear of death or losing control mediates peri-traumatic dissociation and PTSD severity (Gershuny, Cloitre, Otto, 2003). Fear accompanies CP and PTSD (Crombez et al. 1999; Meuller, 2007) and may lead to a cycle of pain and disability through avoidance of certain behaviours, environments (fear avoidance), social and occupational withdrawal, alongside "toxic cognitions" such as catastrophising (among others), and not understanding and confronting their pain (Meuller, 2007; Vlaeyen & Crombez, 1999).

Thoughts and beliefs are nerve impulses, thus imagining a movement can cause pain (Butler & Moseley, 2003) and talking about a traumatic situation can cause hyperarousal (Rothschild, 2000). Thus, emotions perpetuate and reinforce the CP and PTSD experiencing. Memory mechanisms may help explain why traumatic experiences (fear) lead to PTSD (Silva, 2011) and CP (Melzack, 1996; Rome & Rome, 2000).

The left and right hemispheres of the brain give rise to two different systems for processing and recording different types of experience. Different elements of a particular memory are distributed widely across synaptic connections whose values have been shaped and constructed (McClelland, 2011).

Memory is plastic as in each retrieval a memory is edited and changed (reconsolidation) (Silva, 2011; Wilkinson, 2010). As memories change and detail is lost, the emotional core of the event is often maintained (Stickgold, 2011). The role of memory in PTSD is widely known (Mundo, 2006; Ogden, Minton, Pain, 2006; Wilkinson, 2010). It is recognised that treatment involves engaging both the right and left sides of the brain- explicit and implicit relational memory- to allow for maximum integration of the emotional and cognitive experience with the inner and outer worlds (Cozolino, 2002).

CP is processed in the brain areas involved in memory and emotion. Thus "pain memory" (Melzack, 1996; Rome & Rome, 2000) dictates that repeated stimulation of stress and of the emotional parts of the brain from pain perception sensitizes the brain to future pain. Miller (2000) describes CP and PTSD as being neurosensitization syndromes, a complex brain-environmental interaction at many psychobiological levels sharing a pattern of maladaptive positive feedback loops leading to a pathological outcome. Implying neural plasticity in the central nervous system (CNS) at (at least) three different levels. He describes "pain memory", whereby reactivation of a pain memory occurs in response to a peripheral trigger, sometimes months or years after the original memory, suggesting that brain mechanisms may store these pain memories for long periods (Miller, 2000).

Miller (2000) makes no mention of this mechanism within PTSD; however it is implied as a neurosensitization syndrome. Charney, Deutsch, Krystal, Southwick, Davis (1993) elaborated a psychobiological model of PTSD highlighting the role of fear, memory and learning in PTSD.

Emotional memories are implicit and not consciously available; memories about emotions are explicit and conscious (LeDoux & Doyère, 2011). Persistent fear memories are stored in the amygdala, involved in the release of stress hormones (flight/fight/freeze) of the autonomic nervous system. The amygdala displays plasticity and is implicated in fear conditioning (LeDoux & Doyère, 2011); implying that fear can be "unlearnt". However, as remembering renders memory subject to change (emotional updating), fear memories may be strengthened (easier to retrieve and involved in flashbacks/intrusions in PTSD) or weakened during reactivation, through processes undiscovered (LeDoux & Doyère, 2011).

Emotional memory may be useful in learning through stimulus-re-enforcer association learning, whereby an action is performed to avoid the punishment or to obtain the reward (Rolls, 2011). They may be implicit or explicit processes and may be seen in the maintenance of CP and PTSD (avoidance) or in the recovery (to obtain a reward) (Rolls, 2011).

As pain becomes chronic, brain plasticity also occurs through central sensitization. Pain is then perceived by the brain more frequently with reduced stimuli required to initiate the pain neuro-chemical cascade (Butler & Moseley, 2003). One pain message is magnified into many pain messages and the brain perceives more danger than there is. Thus brain responses (movement, thoughts, autonomic and endocrine responses) are now based on faulty information about the health of the tissue at the end of the neurone- a vicious cycle leading to more pain, more central sensitisation, spreading pain and further reducing physical and psychological

functional ability (Butler & Moseley, 2003). Figures 1 and 2 illustrate acute (nociceptive) pain physiology and central sensitization.

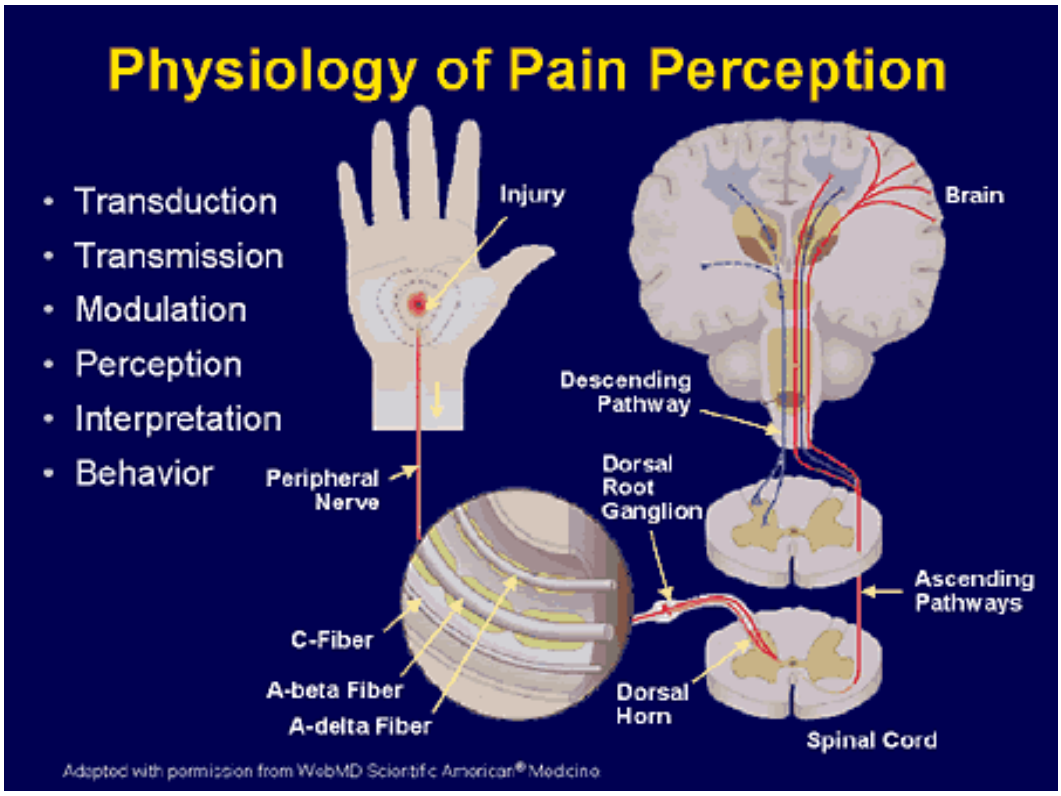


Figure 1: Nociception (from Nicholson, 2004).

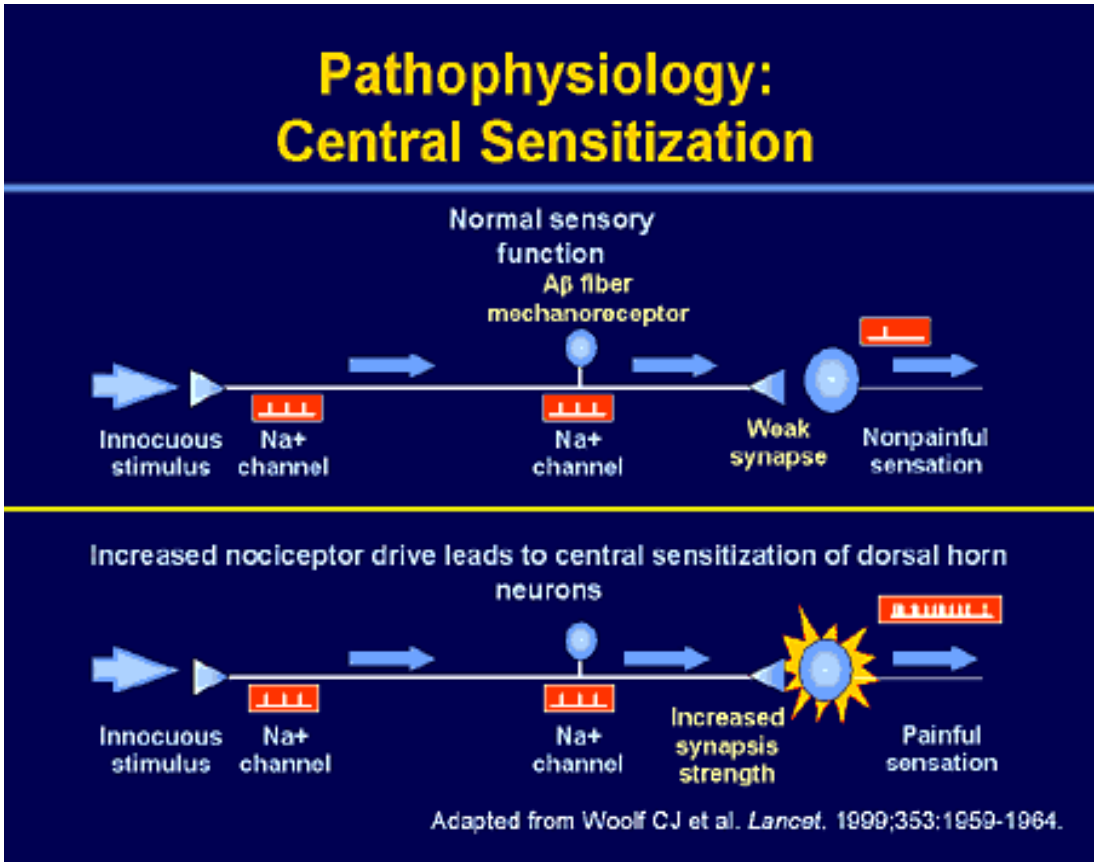


Figure 2: Central sensitization (from Nicholson, 2004).

As pain signals are easier to ignite, pain memory is implicit. Certain thoughts, movements or environments can trigger the release of pain signals and maintain a pain state, flooding the relevant area with pain causing neurotransmitters (Butler & Moseley, 2003).

The brain attempts to protect the body (in a now) maladaptive way- fear avoidance (Kendall, Linton, Main, 1997; Price, 2000). Physical and emotionally threatening events may make the brain more vigilant to threat and thus give it more reason to want to protect the body (Butler & Moseley, 2003). Demonstrating the brain physically changes (plasticity) to maintain and reinforce CP, as the brain responds to experience (Grant, 2011).

Finucane et al. (2012) found significant emotional disturbance in the basic emotions in a group of PTSD individuals; demonstrating pain and emotional memory. They described intense fear or helplessness at time of trauma, with the event often re-experienced on exposure to internal or external cues together with intense psychological distress (Dalgleish & Power, 2004; Gershuny, Cloitre, Otto, 2003; Jovanovic, Norrholm, Fennell, Keyes, Fiallos, Myers, Davis, Duncan, 2009).

CP and PTSD are also stress states (Grant, 1998, 2011; Melzack, 1999) explaining the additional suffering associated with CP including anxiety and depression (Grant, 1998). Stress is involved in pain at all levels of functioning (Grant, 2011) and the subsequent neuroplasticity has been demonstrated on brain scans (in PTSD) (Gerhardt, 2004; Wilkinson, 2010). The prolonged stress making the brain more sensitive to trauma or pain (Gerhardt, 2004; Grant, 2011; Wilkinson, 2010).

These stress mechanisms (HPA-axis) are initiated in PTSD (Gerhardt, 2004; Herman, 1992; Wilkinson, 2010) to “triggers” (Butler & Moseley, 2003; Mersky, 1998, Meuller, 2007) whereby the brain believes there is a threat/danger to the self (Butler & Moseley, 2003; Meuller, 2007). The HPA-axis is initiated in CP to thoughts or movement- a threat to the self (Butler & Moseley, 2003). The sympathetic nervous system (SNS) remains activated in a pain state (fight or flight) releasing adrenaline and cortisol, despite no longer being in the stressful situation. Adrenaline magnifies pain messages generating anxiety and anger; contributing to sleeplessness and the parasympathetic system (PNS) is unable to initiate the “rest and digest” state. The endocrine system is also activated during stress (Butler & Moseley 2003). Pain and stress are inextricably linked (Gatchel, Peng, Peters, Fuchs, Turk, 2007).

PTSD and CP are also characterised by somatic hypervigilance, and hyperarousal associated with detection of pain (Asmundson, Wright, McCreary, Pedlar, 2003); with (possible) biases in attention toward threatening stimuli, and fear avoidance, due to dysregulation in the stress response and pain modulation systems (Asmundson et al. 2002; Gerhardt, 2004; Wilkinson, 2010).

Analgesics in CP have little to no impact on pain perception (Grant, 2011). Since neuroplasticity occurs at many levels (Miller, 2000), administration of a single class of pharmacological agents would, at best, have only a partial and limited effect (Miller, 2000). Psychological therapies therefore are endorsed (Miller, 1997; 1998) as Dubovsky (1997) asserts the therapeutic relationship 'splints' the neurophysiological regulatory mechanisms, providing a repeated corrective stabilization so that the individual can eventually function normally on their own (Miller, 2000).

Since the brain develops structurally and functionally (neuroplasticity) to accommodate maladaptive coping in response to pain or trauma, the brain may also learn to accommodate more helpful ones (Butler & Moseley, 2003; Gerhardt, 2004; Grant, 1998; 2011; Wilkinson, 2010). Reconsolidation-based strategies on memory may therefore provide an opportunity to weaken initially strong memories that result in maladaptive behavioural responses (Silva, 2011). By harnessing neuroplasticity (in the reverse) pain memory, emotional memory, and central sensitization, may be countered (by graded exposure (Butler & Moseley, 2003) among others) to improve functioning and reduce pain. However, as personal and professional experience states, facing ones greatest fears (movement) and its anticipated pain is terrifying.

And despite knowledge of the inter-relationship of mind and body (Grant, 2011) it becomes a leap of faith.

Other interrelated factors to CP include personality, gender, anxiety, depression, coping style, self-efficacy, and locus of control (Butler & Moseley, 2003). These may be characteristics of the individual but there is also the assumption that learning has played a part in their development. As such they are also subject to modification by learning (Butler & Moseley, 2003).

Despite the myriad of correlating factors in CP, pain is the perception of pain itself (Hunter & Lode, 2001) - the brain decides whether something hurts or not (Butler & Moseley, 2003) as the result of stress-induced biochemical, functional and structural abnormalities (Grant, 2011) within it. It follows that the brain is where treatment should be targeted.

Chapter 2 examines EMDR and some psychological treatment options for CP.

CHAPTER TWO.

HOW DOES EMDR WORK IN CHRONIC PAIN?

Chapter 1 highlighted that CP may be viewed as a small-t trauma, with some similarities to PTSD (big-T Trauma) (Grant & Threlfo, 2002), where CP and PTSD diverge is not discussed. Fear, memory, emotions, meaning/context, learning, neuroplasticity and the left and right brain hemispheres are implicated with the maintenance of CP and PTSD. Memory is not fixed, learning new thoughts and behaviours leads to plasticity enabling recovery of movement/environment thereby challenging the previously associated fears. CP and PTSD involve anger and fear (Finucane et al. 2012). Thus interventions should also aim to increase positive emotional experiences, and treat depression where necessary in CP (Grant, 2011). A counselling approach would need to embrace the elements of emotion, learning and memory simultaneously (Burn, Sinel, Deardorff, 2007).

NICE recommends trauma-focussed CBT or EMDR for the management of PTSD in primary/secondary care (NICE, 2005, CG26, 1.9.1.8; 1.9.2.8). It states that non-trauma focused interventions (including non-directive therapy) that do not address traumatic memories should not routinely be offered to those with PTSD due to lack of "convincing evidence" in their efficacy and effectiveness (NICE, 2005, CG26, 1.91.8).

For chronic lower back pain, NICE (2009, CG88) recommends a behavioural cognitive approach to psychological treatment in combination with physical activity and education. Non-directive counselling appears not to have been considered.

In searching the literature (beyond the scope of this dissertation), one noted that PTSD and CP may be managed using the same modalities such as: mindfulness, behavioural, cognitive, hypnosis, and psycho-physiological (biofeedback and relaxation) approaches. Accepting the similarities, it may follow that what potentially works in CP would work in PTSD and vice versa.

CBT prioritizes behavioural and cognitive techniques to control emotion and improve coping abilities (Mazzola, Calcagno, Goicochea, Pueyrredon, Leston, Salvat (2009). CBT views CP primarily as a product of negative thoughts, behaviours and feelings exacerbating perceived pain, rather than physiological pathology (Grant, 1998). Treatment is aimed at reducing pain by changing negative thoughts and beliefs.

Various problems have been noted with CBT treatment and research in CP (Bradley, Young, Anderson, Turner, Agudelo, McDaniel, Pisko, Semble, Morgan, 1987). With high drop out and relapse rates (Turk & Rudy, 1991) suggesting effects are not well maintained. CBT is further challenged by Craig (1999) emphasising the recognition of the role of emotion in CP. Although CBT challenges thinking, emotional, behavioural and physical reactions are also incorporated in therapy work (Greenberger & Padesky, 1995).

Ultimately, ones sense of self has been challenged by CP or PTSD creating incongruence. Person-centred theory states behaviour is goal directed by the individual to maintain or enhance him/herself in response to the experienced environmental conditions (a growth tendency) (Merry, 2004). A person-centred approach to CP and PTSD would seek to establish the psychological environment in

which the individual's actualising process may be expressed positively (Merry, 2004) enabling the individual to harness their inner resources, integrating the incongruence into a new self-concept through exploration of emotions (Gillon, 2007). Influential emotional factors in CP and PTSD have been discussed (Chapter 1) however there is limited evidence in support of person-centred counselling (PCC) within CP or PTSD.

EMDR is a rapid information-processing therapy in which the individual reprocesses traumatic or dysfunctional thoughts, feelings and somatic perceptions (Mazzola et al. 2009). It was originally developed for individuals who had experienced psychological trauma by Shapiro (1989) with demonstrable success in treating PTSD (Carlson, Chemtob, Runsnak, 1998; Maxfield & Hyer, 2002; McCann, 1992; Wilson, Becker, Tinker, 1995)

EMDR is informed by Shapiro's Adaptive Information Processing (AIP) model (2001; 2002) which rests on the idea that when distressing memories are stored in isolation and inadequately processed, the dysfunctional emotions, perceptions and sensations of the initial event remain essentially unchanged. EMDR (for PTSD) consists of an 8-phase process: History, Assessment, Preparation, Desensitization, Installation, Body-scan, Closure, Re-evaluation. Desensitization (dual attention stimulus/bilateral stimulation) enables the individual to review their trauma in a transformative way (Grant, 1998).

The aim of EMDR is to process the incident and the additional memories (loss of function, role, self-worth, identity, shame, anger *et cetera*). These identified targets

are necessary for a permanent elimination of pain, alongside present triggers and future positive templates (Grant, 1998; van Rood & de Roos, 2009).

EMDR facilitates change in affective, cognitive and neurological processes (Grant, 1998). A theoretical effectiveness of EMDR in CP (Grant & Threlfo, 2002) is suggested based upon the neurobiological similarities found in individuals with CP and PTSD (Grant, 1998; van der Kolk, 1994, van der Kolk, McFarlane, Weisaeth, 1995); the co-occurrence of PTSD and CP (Asmundson & Katz, 2009; Beck & Clapp, 2011; Roth, Geisser, Bates, 2008); and that CP itself is a small-t trauma due to the major life-changing events typically associated with CP (loss of income, loss of self, role, function, self-esteem *et cetera*) (Grant & Threlfo, 2002). The emotional focus of EMDR appears congruent with the neurophysiological mechanisms of pain; specifically Rome and Rome's LAPS (2000) with significant reduction in disturbing feelings and sensations (McCann, 1992; Shapiro, 1989; Wilson, Silver, Foster, 1996).

EMDR and dual attention stimulus have been shown to reduce physical and emotional distress associated with memory, and promote relaxation and rapid eye movement sleep. Brain scanning also demonstrated changes to physical and neurological activity associated with painful memories (Levin, Lazrove, van der Kolk, 1999). However EMDR does not appear to allow individuals to explore their experiencing of CP in the way PCC would, rather focussing on facilitating expression of problematic emotional responses in a controlled way to eliminate distressing symptoms. Although it is claimed that EMDR facilitates the individual to identify inner resources that can provide relief (Mazzola et al. 2009), akin to PCC.

EMDR claims to work in tandem with the flow of information to the nervous system in a “bottom-up” direction (Grant, 2011). Simply, sensory data (via brain stem) is relayed to the emotional brain (amygdala) where it is initially appraised before being relayed to the neocortex where the data is more comprehensively analysed (Grant, 2011). The top half of the brain moderates the impact of sensory-emotional data (through suppression or expression) sending signals for action to the amygdala and body (Grant, 2011). Figure 3 illustrates this process.

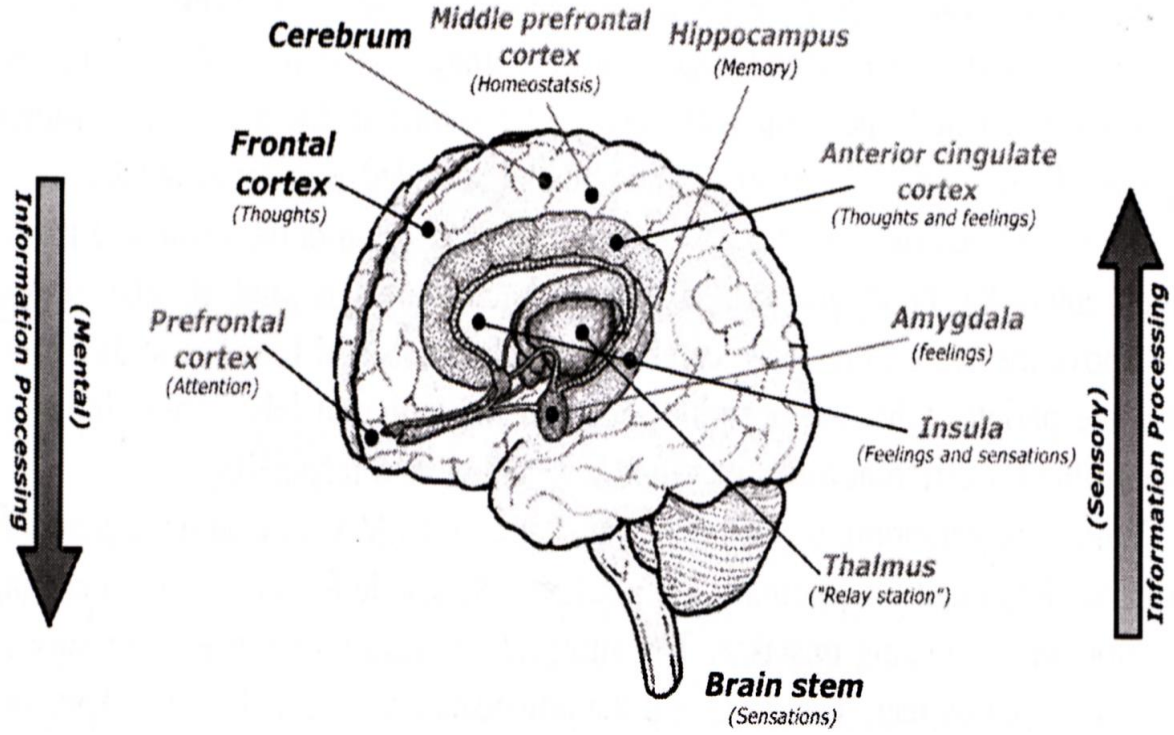


Figure 3: To show the directions of information processing and brain structures (from Grant, 2011, p. 36). Grant (2011) claims EMDR follows a “bottom-up” (sensory) approach whereas CBT and talk-therapies follow a (mental) top-down approach.

In pain protocol (PP) EMDR (Grant, 1998; 2011) for CP, the focus is on the present pain itself, “a present threat rather than a past memory” (Grant, 1998, p. 45). PP EMDR is split into 5 phases to target the effects of stress that maintain CP, and

works similarly to how the nervous system processes information from the sensory aspects of pain towards the cognitive:

- Safety and support
- Reconnecting with your feelings
- Learning how to control stressful feelings and pain
- Changing your thinking
- Building resilience (Grant, 1998, 2011).

Grant (2011) describes talk-therapy (in which I categorise PCT) as following a "top-down" direction of information processing and thus not effective in CP. However I argue that PCT in addressing affect follows a (theoretically) "bottom-up" sensory direction like EMDR.

Roger's non-directive approach (PCC) with World War II veterans (Rogers & Wallen, 1946) is absent from the PTSD literature (Quinn, 2008). Implicit within PCC is the belief that the individual already possesses the resources necessary to become a person. The role of the PCC is as companion/guide in helping the individual navigate their way through (Quinn, 2008). There may be theoretical arguments in proposing PCC in tandem with other modalities such as EMDR or CBT in CP management - beyond the scope of this dissertation.

There appears much to support the theory that EMDR would be useful in treating CP. Chapter 3 discusses methodological considerations in answering the research question:

Can CP be effectively treated using EMDR?

CHAPTER THREE.

METHODOLOGY

Design methods and their rationale for use

Within health and social care, evidence-based practice (EBP) evolved to challenge unjustified variations in clinical practice, protecting the public and the practitioner (Sheldon, 2005). EBP has since spread to all UK public services (Jesson, Matheson, Lacey, 2011). Systematic reviews (SR) provide robust, reliable summaries facilitating decision-making for policy-makers and practitioners, supporting and developing practice and professional development (Dixon-woods et al. 2005).

SR is secondary research (Torgerson, 2003) used to review any methodology of primary research either singularly or mixed methodology (Muir Gray, 2001; Plowright, 2011). Burns (2005), Ham (2005), Lavis, Davies, Oxman, Denis, Bidle, and Ferlie (2005), and Yeates (2005) found managers and policy-makers implementing SR findings found the quality of its conduct (open and reproducible methods) important factors for use. SR is more reliable than a single piece of evidence (Muir Gray, 2001) as acting on the results of a single study is misrepresentative of the balance of available research (Jesson et al. 2011).

SR aims to comprehensively identify all relevant studies to answer a particular question (Glasziou, Irwig, Bain and Colditz 2001; Petticrew & Roberts, 2006) according to a rigorous and pre-defined methodology ensuring an explicit and reproducible methodology (Greenhalgh, 2001). Not all forms of evidence are as equally authoritative (hierarchies of evidence), (Greenhalgh, 2001; Sheldon, 2005)

thus each study is assessed for validity (Petticrew & Roberts, 2006), and explicit methods limit bias in identifying and rejecting studies (Greenhalgh, 2001).

The results of several studies are formally compared to establish generalisability of findings and consistency (lack of heterogeneity) of results (Greenhalgh, 2001). Reasons for heterogeneity can be identified and new hypotheses generated (Greenhalgh, 2001). Conclusions are more reliable and accurate enabling large amounts of information to be assimilated quickly (Greenhalgh, 2001).

Petticrew and Roberts (2006, p. 27) identify seven stages in a SR:

- Define the research question
- Determine the types of studies needed to answer the question
- Undertake comprehensive literature search
- Screen the results against inclusion/exclusion criteria
- Critically appraise the selected studies
- Synthesize the studies, assess heterogeneity
- Disseminate the findings.

A SR was considered appropriate as it could answer the research question:

Can CP be effectively treated using EMDR?

It is a useful methodology when one is uncertain about what evidence is available, or if the intervention is effective (Petticrew & Roberts, 2006). CP and EMDR are clearly defined facilitating a focused SR (Petticrew & Roberts, 2006). A narrow focus

answers the specific question (Petticrew & Roberts, 2006). This SR identified a new/emerging development (and gaps in knowledge) (Dixon-woods et al. 2005).

Philosophical perspective

Regardless of the methodologies reviewed, SR is a positivist approach, adopting operationalization of terms and following a transparent scientific process (Jesson et al. 2011) to limit variables and confounding results, maintaining objectivity, rigour and validity (Greenhalgh, 2001; Petticrew & Roberts, 2006). The research question (seeking effectiveness of an intervention) dictated quantitative data, concerned with measurement, ignoring the human experience (Wilson & MacLean, 2011). Quantitative research is also positivist, a philosophy proclaiming the suitability of scientific method to all forms of knowledge (with a prescribed method) (Bryman, 1988).

Twelve versions of positivism exist (Bryman, 1988) based on the assumption that the methods/procedures of the natural sciences are appropriate to studying social sciences (Bryman, 1988). Thus only phenomena directly observable (through experience or observation), or indirectly using instruments, (observable facts (Delanty & Strydom, 2003)) are warranted as knowledge (empiricism) (Bryman, 1988) without preconception of how they are ordered or what explains them (Coolican, 2009). These empirically established regularities of observable facts are “laws” (induction) (Bryman, 1988; Coolican, 2009; Gray, 2009).

Hypothesis testing (deduction) occurs through empirical observation/experimentation. Requiring (pre-experimentation) operationalizing of underlying concepts, and creating

measurement tools (for observed phenomenon) to confirm what occurred (Gray, 2009). Hence Scientists aspire to be value-free (objective) maintaining validity of knowledge (Bryman, 1988) facilitating the potential replication of studies (demonstrating rigor, reliability, validity). Popper's (1959) hypothetico-deductive method states falsifying a theory is more powerful evidence than proving it (Bowling, 2002). Positivist research may be inductive discovery, deductive proof or both (Dewey, 1933); no positivist branch entails all the above (Bryman, 1988).

The objectivity of positivism is questioned as one rarely observes without preconceptions or assumptions (Coolican, 2009). Positivism dismisses subjective intangible evidence (Gray, 2009) as it cannot be operationalized (Bryman, 1988). As such it is argued that quantitative data and positivism are considered inappropriate for studying people and social reality (Bryman, 1988; Coolican, 2009). Furthermore, deductive research does not answer the "why" of the investigated behaviours (Coolican, 2009).

The hypothetico-deductive method is dependent upon potentially inaccurate observations due to measurement difficulties (Bowling, 2002) as the concept of operationalism may be limiting and misleading (Bowling, 2002). The operational tool may not measure what it purports to (validity) (Bowling, 2002) and standardised procedures may not be followed (Coolican, 2009). Since procedures do not tell the experimenter exactly how to interact with study subjects there is potential for bias (Coolican, 2009). Despite the strict positivist controls, hypotheses are not usually completely supported or refuted by research data (Bowling, 2002).

In practice, science is based on a more fluid blending of deductive and inductive or probabilistic reasoning alongside paradigm shifts extending science flexibly (Bowling, 2002). The idea that CP and PTSD were linked began inductively before this dissertation commenced. In starting this dissertation, I had already formed two hypotheses: 1) CP was a type of trauma; 2) EMDR would be a useful treatment; a deductive approach. If my theory does not fit the data a new theory will emerge inductively (Bowling, 2002).

Despite being empirically distanced from the research, I remain inextricably linked. Husserl broke away from the positivist stance, developing his phenomenological philosophy (Moustakas, 1990). This includes “epoche”, “a kind of detachment from the phenomenon being investigated” (Moustakas, 1990, p.38). The extent to which one can remain totally detached from their area of interest is contentious (even with operationalism) and living with the phenomenon under investigation (CP) brings added tensions.

Critical exploration of reliability, validity, trustworthiness

Jesson et al. (2011) state an understanding or working knowledge of the field is necessary for undertaking a SR. Mulrow (1994) demonstrated experts in a particular field are less likely to provide an objective review of all the available evidence, than a non-expert who approaches the literature with “unbiased eyes”. This view is supported by Oxman and Guyatt (1993) and is personally and professionally relevant to me. I need to be aware of my personal biases, make them explicit and conduct the research as objectively as possible (Bowling, 2002). My bias is a preconceived idea that EMDR would be useful in CP.

Following SR guidelines (Petticrew & Roberts, 2006)- aiming to maintain objectivity, reliability and validity through the reduction of variables which may impact on results (Wilson & MacLean, 2011)- all reviewed papers must satisfy the inclusion and exclusion criteria regardless of their findings and how they may fit with my pre-conceived ideas of what the results may be (Greenhalgh, 2001).

The choice of inclusion/exclusion criteria are made by me, guided by the review question, and theoretical considerations (Petticrew & Roberts, 2006). Publication bias and language bias are inherent threats. SR quality is influenced by effectiveness of the electronic databases used (Jesson et al. 2011) to search for data. A narrow SR focus may not be generalizable to multiple settings or populations and is at risk of resulting in biased conclusions if I narrow the inclusion criteria in such a way as to exclude studies which are in conflict with my beliefs (Petticrew & Roberts, 2006). Thus changes to inclusion/exclusion criteria are noted at the start of the review and records kept of changes made.

Adhering to research critical appraisal criteria (Centre for Reviews and Dissemination (CRD) 2009; Greenhalgh, 2001) (see further section) with documentation of how and why decisions were made throughout the study process (CRD, 2009) enables research findings to be recognised as credible and trustworthy (rigor) (Bowling, 2002; Bryman,1988; CRD, 2009; Gray,2009; Paterson, Thorne, Canam, Jillings, 2001; Petticrew & Roberts, 2006).

Uncontrolled studies are more susceptible to bias than studies with control groups, as without a control group it is difficult to know what would have happened in the

absence of the intervention, making it difficult to be sure that any change in outcome was due to the intervention or some other factor (Petticrew & Roberts, 2006). Systematically reviewing a number of similar uncontrolled studies will not necessarily allow a definitive attribution of causality but will allow consistency of findings among the studies to be explored, providing indicative evidence that the intervention is having an effect (Petticrew & Roberts, 2006).

Operational tools are reliable if it produces the same scores from the same people at different times. Validity demonstrates or measures what the researcher claims it does. A valid measure may have low reliability (Coolican, 1990). Valid operational tools within primary studies adds rigor to the SR findings (Bowling, 2002). Threats to reliability, validity, and trustworthiness arise in the way I conduct the SR and also from the primary studies themselves. SRs are usually restricted to published, peer-reviewed, academic work to improve rigor (Jesson et al. 2011) yet describes publication bias (Petticrew & Roberts, 2006). Due to lack of research my search was widened to include non-peer review published academic work. This may impact upon the SR results and ability to locate papers from less mainstream sources. Language bias results from searching only English language papers.

SRs are usually a team effort to reduce bias. As a lone researcher, to maintain trustworthiness, validity and reliability, I follow the frameworks rigidly providing a replicable audit trail (Petticrew & Roberts, 2006).

Ethical considerations

No ethical approval was required for this SR as the primary studies have sought ethical approval. My research conduct and integrity– from the literature searches, handling of the data and the analysis- are ethically bound (British Association for Counselling and Psychotherapy (BACP), 2004). I have an ethical duty of care to myself, the represented authors, and to my clients (BACP, 2004). Furthermore, I have an ethical consideration to my peers, colleagues and the counselling profession (Etherington, 2004).

An ethical expectation is that research results are disseminated to valid interested parties (Bowling, 2002; Jesson et al. 2011; Petticrew & Roberts, 2006). I will allow this dissertation to be stored in the university library, and seek publication within the counselling and CP communities. I will also email the authors I previously contacted (see section further) to enquire whether they would like a copy of this dissertation upon completion (Grant has affirmed he would).

Aside from a university requirement, there is an obligation within the BACP Ethical Framework (BACP, 2010) to participate in research, maintain competent practice, share research findings, impart knowledge and act in the client's best interests. This dissertation satisfies these obligations and has clarified where my future research and practice areas may be.

Inclusion and exclusion criteria for primary studies

Inclusion criteria should capture all studies of interest. Criteria too narrowly defined risks missing potentially relevant studies; reducing generalizability of results. Too broad, makes comparison and synthesis difficult impacting upon rigour (CRD, 2009).

As an emerging area of enquiry and investigation, literature directly related to my research questions is extremely limited. My searches were therefore modified (where specified in the following inclusion/exclusion criteria).

Inclusion criteria:

- Text directly relating to the dissertation title
- Initially I searched only peer reviewed journals, due to lack of results I broadened the search to include non-peer reviewed journals
- Intervention studies (case studies, controlled and uncontrolled clinical trials)
- Literature reviews/synthesis/analysis
- Adult participants, 18 years plus, as I only work with adults
- CP is defined as non-malignant chronic musculo-skeletal pain (with or without a medical diagnosis). Extended to include migraine/headache, fibromyalgia, somatoform, neuropathic and phantom limb pain due to lack of results
- To ensure no data was omitted the search was from 1989 (when Shapiro published her first article on EMDR) to August 2012.

Exclusion criteria:

- Papers not written in English, due to the potential for loss of meaning in translation (potential for language bias)

- Where CP is part of an on-going disease process such as multiple sclerosis or arthritis to reduce variables of disease processes/progression upon results
- Veterans with CP and PTSD. I am aware that all CP has the potential to have arisen from a traumatic event; this is an attempt to look at CP separate from PTSD
- Studies investigating EMDR with non-pain conditions
- Conference papers and posters, due to difficulty/inability to obtain them within the dissertation timeframe (publication bias).

Identification and selection of studies

A three-step search strategy is recommended for systematic reviews (van Rood & de Roos, 2009); employed here to increase rigour and to ensure papers were not missed.

STEP 1. I searched the University library computerised databases (Cinhal plus, Medline, PsycARTICLES, PsycBOOKS, Psychology and Behavioural Sciences Collection, PsycINFO). I used broad keywords in combination with Boolean terms as a preliminary assessment of available literature: **"EMDR" AND "Chronic pain"**. Limitations set: human, adult, English language, 1989-2012 (start date appropriate as that was when EMDR was first discovered by Shapiro).

This returned seven results (Appendix II). Of these, three were relevant (Grant, 2000; Grant & Threlfo, 2002; Mazzola et al. 2009). Four were excluded: one concerned single session EMDR with hypnosis (Ray & Page, 2000); one was an editorial (Shapiro, 2002a); two concerned phantom limb pain (de Roos, Veenstra, de

Jongh, den Hollander-Gijsman, van der Wee, Zitman, van Rood, 2010; Schneider, Hofmann, Rost, Shapiro, 2008).

To avoid repeating an earlier SR, I searched for existing reviews (Petticrew & Roberts, 2006) using the Cochrane Database (www.thecochranelibrary.com) and DARE database (www.york.ac.uk/inst/crd) using the above search terms, returning no results.

STEP 2. Reference sections of the three relevant papers were inspected for relevant studies that had not yet been detected (back-chaining), generating one new paper (Hekmat, Groth, Rogers, 1994) and two books which I purchased (Grant, 1998; 2011). Hekmat et al. (1994) concerned laboratory induced acute pain and were therefore not applicable; back-chaining from it generated no results. Back-chaining the books (Grant, 1998; 2011) generated no new results.

STEP 3. The two lead authors (Grant, was lead author in two papers) of the three selected articles (Grant and Mazzola) were emailed asking them whether they had articles on this topic submitted for publication. One author (Grant) responded with a systematic review (van Rood & de Roos, 2009) later eliminated since it included phantom limb pain, and non-pain conditions (psychogenic non-epileptic seizures, stress-related dermatological disorders, chronic fatigue syndrome, and olfactory reference syndrome). The remaining author (Mazzola) has not responded.

I searched the EMDR International Association (www.emdria.org, accessed August 2012) article archive from 1989 to August 2012 for relevant titles/abstracts. This

generated one new paper (Hassard, 1995) which was not relevant as subjects were treated for traumatised memories/flashbacks.

On reading the three relevant primary studies I felt I did not have enough evidence to perform a SR. Closer inspection of Mazzola et al. (2009) revealed the use of subjects with headache, fibromyalgia and neuropathic pain. I was therefore unable to justify these conditions in the exclusion criteria. Furthermore the case study of "Tanya" in Grant (2000) is the same "Tanya" in Grant and Threlfo (2002), reducing the evidence-base further.

Unable to perform a SR with or without the 38 subjects in Mazzola et al. (2009) I decided to widen the inclusion criteria to include pain complaints such as: headache/migraine, neuropathic pain, phantom limb pain and fibromyalgia, to ensure all available papers were located.

The inclusion/exclusion criteria (previous section) were refined to accommodate this addition, other limitations remained unchanged. I undertook new searches (listed below) to reflect the widened inclusion criteria. "somatof*" is included to allow for variations on "somatoform" in the results, as the CP conditions under review are often labelled as somatoform pain (Bacon, Bacon, Hampton Atkinson, Slater, Patterson, Grant, Garfin, 1994; Ciccone, Just, Bandilla, 1996).

EMDR AND headache: generated 2 papers (Appendix II) both relevant (Konuck, Epozdemir, Atceken, Aydin, Yurtsever, 2011; Marcus, 2008). Konuck et al. (2011) was unable to be located through the British Library (Appendix II). At time of writing

(October 2012) Marcus (2008) has not been located by the British Library. At the writing-up deadline and unable to wait I have excluded this paper.

EMDR AND migraine: generated the above two papers.

EMDR AND fibromyalgia: generated no results.

EMDR AND somatof*: generated 2 papers (Appendix II) not relevant- concerning a non-pain condition (Kelley & Benbadis, 2007), and PTSD (Hogberg, Pagani, Sundin, Soares, Aberg-Wistedt, Tarnell, Hallstrom, 2007).

EMDR AND phantom limb pain: generated five papers de Roos, Veenstra, de Jongh, den Hollander-Gijsman, van der Wee, Zitman, van Rood (2010), Schneider et al. (2008) previously returned in Step 1, plus Schneider, Hofmann, Rost, Shapiro (2007). Two were excluded as concerned veterans and PTSD with CP (Russell, 2008a; Silver, Rogers, Russell, 2008).

To reflect the widened CP definition, Step 2 was repeated in all the relevant papers revealing Wilensky (2006). Within back-chaining the previously discarded paper, van Rood & de Roos, (2009), a relevant paper was identified (Friedberg, 2004) and Russell (2008a; 2008b) was discarded as concerning veterans. From Friedberg (2004), back-chaining generated no new papers.

At time of writing (October, 2012), no author (other than Grant) has responded to my email asking if they had any unpublished papers.

Tables 1 and 2 in Chapter 4 show the included and excluded studies.

Critical review and appraisal of papers

An initial screening of titles and abstracts against the inclusion/exclusion criteria identified eight potentially relevant papers which were more critically assessed (Appendix III).

Pawson, Greenhalgh, Harvey, Waishe (2005) ask the researcher to use their own judgement when considering the paper's "fitness for purpose" (p. 24) alongside the use of formal critical appraisal checklists. However CRD (2009) do not recommend their use as they lack reliability and validity (Moher, Jadad, Nichol, Penman, Tugwell, Walsh, 1995).

Nevertheless, the quality of included studies impacts upon reliability of the results and conclusions (CRD, 2009). The following (from CRD, 2009) were considered in assessing quality:

- Appropriateness of study design to the research objective
- Risk of bias (internal validity)
- Other issues related to quality
- Choice of outcome measure
- Statistical issues
- Quality of reporting
- Quality of the intervention
- Generalisability (external validity).

Greenhalgh (2001) adds the extent to which the design and conduct of the study are likely to have prevented systematic errors (bias), and the extent to which the results are generalizable to a particular target population.

Using only English Language studies introduces language bias to the review (CRD, 2009). Publication Bias is reduced in the study design as I have not restricted my literature search only to peer review journals (CRD, 2009). Unpublished studies and "grey literature" are harder to source and more difficult to obtain (Greenhalgh, 2001) than published studies and I am working to a tight timeframe. However no unpublished papers were offered from the contacted authors. I was unable to locate all papers identified from the search (see previous section).

Data analysis

Petticrew and Roberts (2006) describe the process once relevant papers have been located and appraised:

- Organise a description of studies into categories
- Analyse findings within each category
- Synthesise findings across all included studies.

Following the above process, the results are presented in Chapter 4.

CHAPTER FOUR.

FINDINGS

Types of studies

The eight studies (Table 1, arranged to study type then chronologically) consisted of two case studies (Grant, 2000; Schneider et al. 2007) four case series (Grant & Threlfo, 2002; Wilensky 2006; Friedberg, 2004; Schneider et al. 2008), and two uncontrolled clinical studies (Mazzola et al. 2009; de Roos et al. (2010). In total 68 (excluding two duplicated) individuals received EMDR for a CP complaint. Two subjects ("Tanya" from Grant, 2000; "Tom" from Schneider et al. 2008) are excluded as being duplicated in ("Tanya") Grant and Threlfo (2002) and ("Tom") Schneider et al. (2007).

Study designs

All studies used a pre-test/post-test design. Follow up data was collected in all but one case study (Grant, 2000), in three out of five individuals within a case series (Wilensky, 2006) and an uncontrolled clinical study (*n*=38) (Mazzola et al. 2009). The period of follow up measurements ranged from 2-3 months (Grant & Threlfo, 2002, Friedberg, 2004) to 1-2 years (Schneider et al. 2007; 2008; Grant & Threlfo, 2002; Wilensky, 2006) to 2-3 years (de Roos et al. 2010) after treatment.

Participants

The gender of all subjects was presented in all studies except Schneider et al. 2008 (whom it is reported by van Rood & Roos, 2009 from a personal communication that four of the five were male). Of the total sample 48 subjects were female, 21 male.

Mazzola et al. (2009) did not report the age of their participants. The age of the remaining 16 women and 15 men ranges from 25-67 years. Duration of CP ranges from 1 week to 20 years. Note: at 1 week pain is not classed as chronic (IASP, 1986). The type of complaint studied was diverse, including fibromyalgia, phantom limb pain (PLP), neuropathic pain and chronic pain, with and without a traumatic element.

Table 1. Characteristics of the studies included in this review

(Following page).

Table 1. Characteristics of the studies included in this review

Author	Study	*n*	Somatic symptom	Duration of somatic symptom	Number of sessions & protocol used	Follow-up	Outcome
Grant, 2000	Case	1	Leg pain (from accident)	2 years	2 x Pain Protocol (Grant, 1999) ? session length	-	Pain-free days
Schneider et al. 2007	Case	1	Phantom Limb Pain (PLP) (from accident) leg & part of pelvis	3 years	9 x Standard EMDR protocol ? session length	3 months, 1 year & 18 months	Pain free
Grant & Threlfo, 2002	Case series	3	1. Back, neck & hip (accident) 2. Hip (accident) 3. Jaw, neck, shoulder, arm	1. 4 years 2. 10 years 3. 2 years	9 x Pain protocol (Grant, 1999) hourly sessions	2 months & one subject at 2 years	Pain reduced in all subjects, ability to reduce pain improved in all, ability to control pain improved in all
Wilensky, 2006	Case series	5	1. PLP foot 2. PLP foot 3. PLP arm 4. PLP finger 5. PLP leg	1. 3 months 2. 2 months 3. 3 years 4. 5 months 5. 1 week	1. 5 x Standard 2. 3 x Standard 3. 9 x Standard 4. 8 x Standard 5. 3 x Standard protocol ? session length	1. - 2. – 3. – 4. 1 year 5. 3 years	Pain reduced in all and maintained at F/up
Friedberg, 2004	Case series (pilot study)	6	Fibromyalgia & chronic fatigue syndrome	1. 8 years 2. 7 years 3. 5 years 4. 4 years 5. 20 years 6. 17 years	2 x 60minutes EMD plus 2 x 10minutes self-administered EMD at home daily. Authors EMD protocol	3 months	Pain reduced in all

Schneider at al. 2008	Case series	4	1. PLP leg 2. PLP forearm 3. PLP leg 4. PLP leg	1. 4 years 2. 5 years 3. 10 years 4. 16 years	1. 15 x Standard protocol of 50 minutes 2. 4 x Standard 90 minutes 3. 4 x Standard 90 minutes 4. 3 x Standard 90 minutes	1. 24 months 2. 14 months 3. 14 months 4. 21 months	Pain reduced in all and maintained at F/up
Mazzola et al. 2009	Uncontrolled clinical study	38	Headache = 30 Fibromyalgia = 4 Neuropathic pain = 4	Average 12 years	12 x Pain protocol (Grant, 1999) 90 minute sessions. Relaxation & visualization techniques for distress at home	-	Statistically significant pain reduction
deRoos et al. 2010	Uncontrolled clinical study	10	PLP leg	At least 12 months with severe disabling pain for at least 5 days a week	Ranged 3-10 x Standard protocol (trauma memory & pain memory). Standard protocol /pain protocol hybrid (based on Grant, 1999) for in-session PLP. End criteria fulfilment directed session number	26-40 months (mean 2.8 years)	Statistically significant pain reduction maintained at F/up

Table 2. Characteristics of the studies excluded from this review

Authors	Reason for exclusion
Hekmat et al. 1994	Laboratory induced acute pain and EMDR in healthy subjects
Hassard, 1995	Treating flashbacks in a CP population
Ray & Page, 2002	Hypnosis and EMDR in a single session
Shapiro, 2002a	Editorial for journal -describing papers within journal
Hogberg et al. 2007	PTSD in public transport workers
Kelley & Benbadis, 2007	Non-epileptic seizures. Not related to CP
Marcus, 2008	Waited over a month for paper, unable to wait longer
Russell, 2008a	Traumatic amputation related phantom limb pain. Veteran.
Russell, 2008b	Veterans
Silver et al. 2008	Veterans
van Rood & deRoos, 2009	Systematic review including studies concerned with non-pain conditions
Konuk et al. 2011	Unable to be located from British Library

Tables 4-11 (Appendix III) present a critical overview of the primary studies reviewed.

Comorbid psychiatric disorders

Four studies conducted structured clinical interviews for Axis I or Axis II disorders (Friedberg, 2004; Schneider et al. 2007; Mazzola et al. 2009; de Roos et al. 2010).

Protocol

In all studies, the rationale for the use of EMDR was stated (pain memory and trauma memory) and using the AIP model for thoughts/feeling which are not integrated or

available for processing and outside of conscious awareness. The neurobiological similarities of CP and PTSD were also commented rationale.

Three studies (Wilensky, 2006; Schneider et al. 2007; 2008) with 10 subjects used the standard EMDR protocol for PTSD (Shapiro, 1995, 2001). Three studies (Grant, 2000; Grant & Threlfo, 2002; Mazzola et al. 2009) with 41 subjects used the EMDR pain protocol (Grant, 1999). de Roos et al. (2010) (*n*=10) used the standard EMDR protocol (for trauma memory and pain memory) and a hybrid version of the EMDR standard and EMDR pain protocol (for in-session pain). One study looked at EMD (Friedberg, 2004) with no protocol identified (*n*=6).

Three studies involved teaching subjects EMDR components for home use (Grant, 2000; Grant & Threlfo, 2002; Mazzola et al. 2009) and Friedberg (2004) involved home use of EMD. The number of EMDR sessions was not given a rationale except by de Roos et al. (2010) who based the number on the subject fulfilling end criteria (not stated). Length/frequency of EMDR sessions was not rationalized.

Results of the studies

Drop outs

Mazzola et al. (2009) report the number of subjects (*n*=12) who dropped out of their study during EMDR treatment (n=38 remaining). Reasons stated as (*n*=4) difficult to miss a working day to attend clinic, (*n*=2) lived too far away, (*n=5*) without explanation, 1 subject, making good progress, postponed treatment after the 5th session until she had obtained her disability pension. Schneider et al. (2008) lost 2 subjects (50%) one was discharged from hospital; the other's insurance company

refused further reimbursement although there appear to be follow-up data. Wilensky (2006) had 2 subjects (from 5) withdraw as they were happy with their reduced pain and improved functioning. Grant and Threlfo (2002) lost 2 subjects at 2 year follow-up (no explanation). De Roos et al. (2010) lost (*n*= 4) at follow-up. Reasons stated as (*n*=1) withdrew after 4th session (no reason), (*n*=1) died from cancer but had shown improvement, (*n*=1) damaged stump prior to follow-up, (*n*=1) did not have time for data collection.

Comorbidity

At least one axis II disorder was identified in the majority (73.7%) of subjects in Mazzola et al. (2009) (with obsessive compulsive disorder most prominent, 44.7%, others not stated), in 100% of Friedberg's (2004) subjects (*n*= 3 generalized anxiety disorder, *n*= 3 dysthymia, *n*= 1 panic disorder) and in Schneider's et al. (2007) case study (PTSD). All subjects fulfilled the criteria for pain disorder using DSM-IV-TR in de Roos et al. (2010) and *n*= 1 for PTSD.

Primary outcome measures

The number of EMDR sessions, duration and frequency are shown in Table 1, demonstrating pain reduction/amelioration occurs rapidly from two weeks (Friedberg, 2004) to 15 weeks (Schneider et al. 2008). The length of time an individual has CP does not appear influential in outcomes.

Analgesia consumption was monitored in Schneider et al. 2007, 2008; Mazzola et al. 2009; de Roos et al. 2010. Opiate consumption reduced from 600mg/day (pre-intervention) to 100mg/day (post- intervention to 100mg as required (rarely taken) in

Schneider et al. (2007). Schneider et al. (2008) report opiate consumption as unchanged (100mg/day) in subject 1, reduced from 180mg/day (pre- intervention) to 100mg/day (post- intervention and follow-up) in subject 3. Subjects 2 and 4, not taking opiates, had unchanged analgesia. Mazzola et al. (2009) report a 79.49% reduction in analgesia (no opiates) in 30 subjects with no change in the remaining 8 from pre- to post- intervention (no follow-up). De Roos et al. (2010) required subjects to maintain their current analgesia schedule (no details) throughout the study.

All studies except Grant (2000) used valid and reliable tools to measure the effect of EMDR on pain intensity. Grant (2000) appears to note subject's self-report of pain but without a "score", pain reduction cannot be objectified. A numerical rating scale, faces scale, or visual analogue score (0-10, 0=no pain, 10=worst) is used by Wilensky (2006); Schneider et al. (2007; 2008); Mazzola et al. (2009) and de Roos et al. (2010). However, 2 of the subjects in Schneider et al. (2008) fail to give a numerical rating, using descriptors.

Subjective Units of Distress (SUD) are used during the EMDR treatment process to measure response to treatment. Friedberg (2004) and Grant and Threlfo (2002) measure pain intensity as part of the SUD (0-100 numerical rating scale, 0=no pain, 100/10= as intense as it could be). This is not consistently reported by Grant and Threlfo (2009) for all subjects. Mazzola et al. (2009) state pain focused SUD scores are obtained but provide no details of scores. Wilensky (2006) and Schneider et al. (2007) use a SUD to measure current level of distress rather than pain. SUD ratings for pain in Friedberg (2004) decreased by (mean) 17.1% in session 1, and 18.5% by end of session 2 (35.6% mean reduction).

The Short-form McGill Pain Questionnaire (SFMPQ) (15 pain descriptors, 0-5, and pain intensity: mild=1, moderate=2, severe=3, max 77) is used by Grant and Threlfo (2002). They report pain reductions by almost half: 44, 32, and 15 (to 28, 34, and 28 out of 77) in each subject from pre-intervention to 2 month follow up. Diaries were also utilized to record pain (with 4 measurements a day (Friedberg, 2004), and 3 measurements a day (de Roos et al. 2010)). Friedberg (2004) used the Fibromyalgia Impac Scale to measure pain alongside other outcomes. Bodily pain is measured through the Short-Form Health Survey (Mazzola et al. 2009; de Roos et al. 2010).

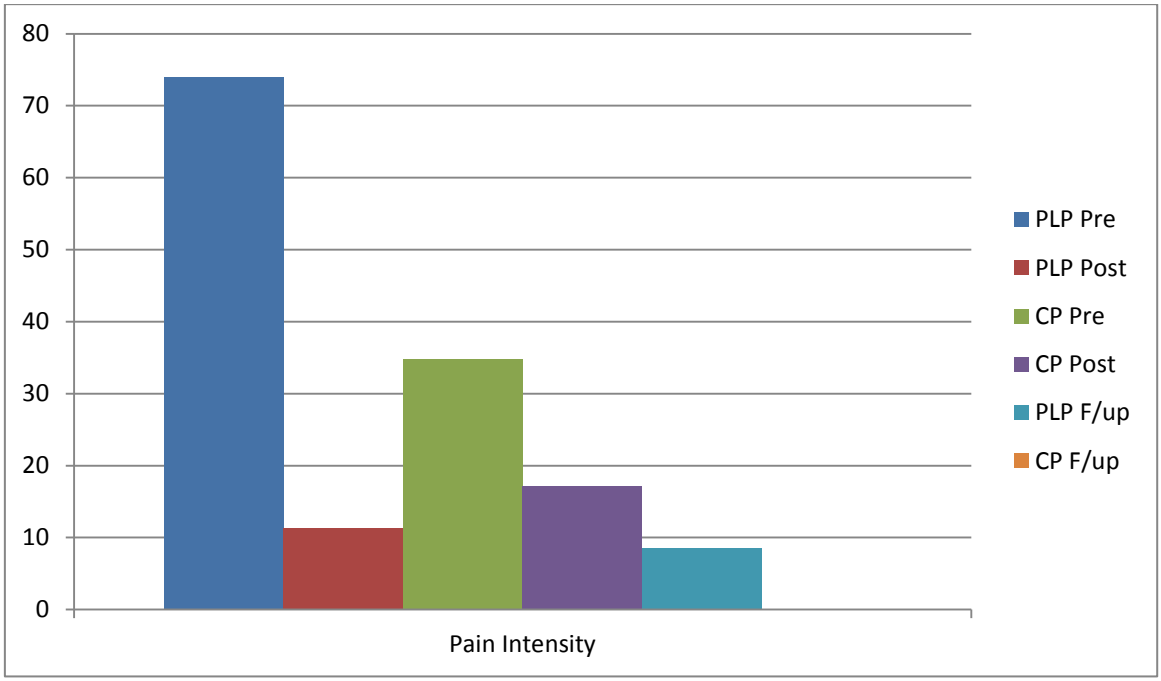
Where comparable numerical data were available, Table 3 shows pre-intervention and post-intervention pain scores, Graphs 1 and 2 show pain scores correlated to pain type and EMDR protocol used (following pages).

Table 3. To show pain scores pre-intervention, post-intervention, and at follow-up, using a numerical rating scale.

Pain type	Author	*n*	Pre-EMDR	Post-EMDR	F/up Score	F/up period
PLP	Wilensky, 2006	5	10, 7, 8, 5, 9 mean SP	0, 1, 1, 1, 0 mean	1, 0 mean	1 year 3 year
PLP	Schneider et al. 2007	1	10 SP	0.5	0	18 mths
PLP	Schneider et al. 2008*	2 (4)	10, 10 SP	0, 5	0, 5	14 mths
CP	Mazzola et al. 2009	38	4-10 median 8 PP	1-9 median 6	-	-
PLP	De Roos et al. 2010	10	5 mean SP/PP	2.8 mean	2.5 mean	26-40mths mean 2.8yr

Key: SP= standard EMDR Protocol PP= pain protocol
*Only 2 subjects (from 4) used numerical data.

Graph 1: To show total pain scores pre- post- EMDR comparing CP to PLP.

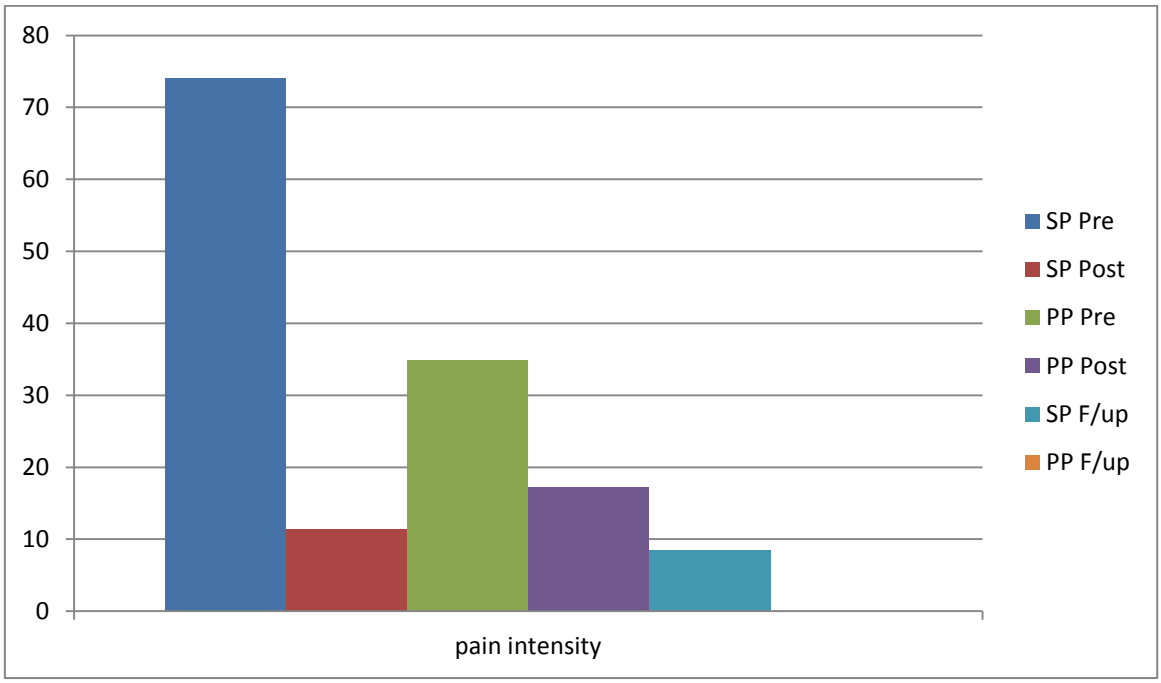


Note: No data available for CP follow up.

Data converted where possible from all studies (Grant & Threlfo, 2002; Friedberg, 2004; Wilensky, 2006; Schneider et al, 2007, 2008; Mazzola et al, 2009; De Roos et al, 2010) onto a 0-10 rating, and added up with totals represented in the graph. Grant (2000) not represented- no numerical data. Data from "Tom" included once (in Schneider et al, 2007).

From this graph, PLP subjects (*n*=18) reported higher pain scores pre-intervention with lower pain scores post intervention (maintained at follow-up). CP subjects (*n*=20) reported less pain intensity with a lesser reduction in post-intervention pain score. No numerical data was available for follow-up in the CP subjects, although treatment gains are maintained at 2 years (Grant & Threlfo, 2002).

Graph 2: To show total pain scores pre- post- EMDR comparing standard EMDR protocol (SP) to Pain Protocol (PP).



Note: No data available for PP follow up.

Data converted where possible from all studies (Grant & Threlfo, 2002; Friedberg, 2004; Wilensky, 2006; Schneider et al, 2007, 2008; Mazzola et al, 2009; De Roos et al, 2010) onto a 0-10 rating, and added up with totals represented in the graph. Grant (2000) not represented- no numerical data. Data from "Tom" included once (in Schneider et al, 2007).

This graph shows that SP (*n*=18) subjects have a higher pre-EMDR pain intensity and a lower post- EMDR pain score, maintained at follow up. PP subjects (*n*=47) reported less pain intensity with a lesser reduction in post-intervention pain score.

The two graphs are identical: the PLP studies used the SP, CP studies used the PP. The de Roos et al. (2010) data (who used SP and a hybrid PP) was put into the SP data as I was unable to place which figures were based on which protocols, and the majority of the protocol used (de Roos et al. 2010) was SP. These graphs indicate that PLP responds better to SP EMDR than CP does to PP EMDR. They may also

indicate that SP is more effective than PP however without any PLP studies using a PP (and vice versa) this cannot be substantiated.

The CP and PLP studies differed in the primary targets used. Trauma-related memory was the primary target of EMDR in the PLP studies (de Roos et al. 2010; Schneider et al. 2007; 2008; Wilensky, 2006) using the SP. Pain sensation as the primary target was seen in the CP studies using PP (Grant & Threlfo, 2002; Mazzola et al. 2009). "Tanya's" data (Grant, 2000) was included in Grant and Threlfo (2002) with more detailed reporting. Friedberg (2004) did not identify a primary target.

Of all the individuals for whom data on comorbidity were available, only two fulfilled the criteria for PTSD according to DSM-IV (-TR) (Schneider et al. 2007; de Roos et al. 2010). Demonstrating EMDR is effective when PTSD is not implicated in the CP condition. However forgotten traumas were realised during the course of EMDR (Wilensky, 2006).

Thinking of the PLP success, perhaps EMDR is more effective where individuals have higher pain intensity levels; when there is trauma memory to target; or where strong emotions are attached to the injury. The CP studies may have less successful results as the actual pain is used as a target due to lack of trauma. In those with traumatic pain, the SP EMDR may have had a greater pain intensity reduction than non-traumatic CP PP EMDR as the SP EMDR facilitated memory processing of the traumatic incident. Highlighting perhaps a difference in memory processing in traumatic compared to non-traumatic CP.

The differences in pain intensity scores between the two groups (PLP and CP) is noteworthy, as where individuals may place themselves may be influenced by pain type, length of pain, gender, coping ability *et cetera*. From my experience, in the acute phase of non-traumatic pain is rated highly (say 8/10). In chronicity, the pain rating generally reduces as one, to greater and lesser extents, integrates CP into daily living with flares-up of pain intensity, becoming a more constant background pain (say 3-4/10), compared to the attention grabbing early pain.

This may explain why the CP subjects had lower pain scores pre-intervention compared to the PLP subjects - the CP PP group may have adapted better to CP over time than the PLP SP individuals. The nature of PLP may also be qualitatively different to other types of CP, influencing results. Looking at the changing pain intensity, frequency and sensation over time in CP and PLP may illustrate more clearly what factors are at play.

All subjects reported symptom improvement. The mixed group of CP individuals is heterogeneous for the type of pain and origin of pain, whereas all the studies on PLP and EMDR included individuals who had lost a limb (in most cases through a traumatic event). If EMDR is more effective in individuals with trauma-related CP, one would expect better results in PLP individuals than a more varied group of CP individuals. Chapter 5 will present a discussion and conclusion.

CHAPTER FIVE.

DISCUSSION AND CONCLUSION

Discussion

Pain is comprised of nociceptive input and emotional reactions that influence the individual's psychological welfare, exacerbate unpleasantness, helplessness, anxiety, depression, pain perception, and pain intensity (Hadjistavropoulos & Hadjistavropoulos, 2000; Rainville, Bao, Chretien, 2005). When pain is chronic, the constant feeling of pain, fatigue, and distress becomes a traumatic experience where the source of danger resides in the body (Shapiro, 1995). Grant and Threlfo (2002) suggest that the (small-t) trauma of CP also arises from loss of self, role, function, inability to work, and family/marital stress among others.

Over the last two decades, our understanding of the modifiability of the primary sensory and motor areas of the brain has grown. It is recognised that plastic changes of the primary cortical areas occur throughout life as a consequence to both injury and stimulation (Flor, 2003). Clinical observations and neurobiological evidence suggests that CP and PTSD share some important similarities (Mazzola et al. 2009) including areas of the central nervous system implicated in the experience of both pain and trauma (Bergman, 1998; Grant & Threlfo, 2002; Price, 1999).

EMDR is an integrative, psychotherapeutic approach (somatic, emotional, cognitive and behavioural) consisting of a desensitizing procedure, alongside other interventions (such as meditation, exposure and relaxation), designed to facilitate a more adaptive cognitive and emotional state (Shapiro, 1998). It stimulates

information processing of distressing memories, thoughts, feelings and somatic perceptions through bilateral stimulation and dual focus of attention (Grant & Threlfo, 2002; Mazzola, et al. 2009). There are various mechanisms by which EMDR is thought to reduce/ameliorate pain which shall be discussed.

Memory and processing of pain and trauma

The AIP Model (Shapiro, 1995; 2001) guiding EMDR states that like traumatic experiences, CP may be the result of unassimilated neurobiologically stored memories related to the source of the pain itself, the long-standing state of pain, medical procedures, or other unresolved distressing events (Bergman, 1998; Flor, 2002; Schneider et al. 2007; Shapiro, 1995; 2001). CP involves the automatic emotional response to pain perception and the somatic component of stored memories (Schneider et al. 2007). Cognitive, perceptual and affective components of CP are intertwined. Changes to the cognitive and/or affective components can initiate modification in sensory discrimination of pain (Price, 1999).

The secretion of stress hormones in severe stress conditions is thought to lead to overconsolidation of traumatic memory storage inhibiting cognitive evaluation and semantic representation of the experience (Le Doux, 1992; 1994; van der Kolk, 1994). Traumatic memories can be stored in the sensori-motor system where sensations and visual images can initiate response memories (Le Doux, 1992; 1994; van der Kolk, 1994). Unresolved traumatic memories may therefore amplify the emotional dimension of the pain experience; emphasizing the importance of psychological interventions to pain management (Mazzola et al. 2009).

EMDR appears to desensitize the automatic emotional response to pain and the somatic component of stored memories related to pain onset. Desensitizing the limbically enhanced portion of the pain experience allows a more normal affective response to pain signals and stressful events (Ray & Zbik, 2001). This disconnecting of traumatic memory and painful associations allows the individual to experience pain with less disturbing feelings and distress (Ray & Zbik, 2001; Shapiro, 1995; 2001). The negative cognitions and feelings are replaced by learning new adaptive strategies to improve their condition (Mazzola et al. 2009).

The results of the PLP studies indicate that EMDR is effective in treating unresolved traumatic and pain related memories in most individuals. de Roos et al. (2010) question from their results if it is necessary for individuals to have an explicit amputation-related memory that is still affect-laden to benefit from EMDR. Only one of their subjects fulfilled the criteria for PTSD, although their sample generally reported more trauma symptoms than the Dutch population. Of the study sample 70% reported trauma or pain-related memories that were affect-laden which were targeted for EMDR. In 3 PLP subjects (from 10) no disturbing pain or trauma memories were identified. Two individuals (treatment non-responders) regarded their amputations and related events as positive life-saving events (de Roos et al. 2010). This may have implications for why CP PP EMDR studies had less apparent successful outcomes; perhaps their pain was not as affect-laden as the PLP subjects.

Long-standing pre-amputation pain appeared to be related to less successful EMDR outcome, suggesting somatosensory memories may depend on a sensitivity

threshold to pain intensity and duration (de Roos et al. 2010). Future studies should contain details of pre-amputation pain duration, intensity and characterization so this link may be explored further (de Roos et al. 2010).

The PP EMDR was not used in PLP. However during the course of SP EMDR, pain sensations did become the focus of treatment as they emerged during the targeting of the initial accident and other specific events (Schneider et al. 2007). Schneider et al. (2007) recommend using the present pain as a target "if processing of past, present and future is not sufficient" (p.42). An extensive follow-up is necessary to identify new triggers that may elicit unresolved issues or cause re-traumatization (Schneider et al. 2007).

The interaction between interhemispheric and intrahemispheric interactions (lateralization hypothesis) during pain processing is also demonstrated by EMDR. Right cerebral activation is reported in response to noxious stimuli and appears related to the right hemisphere's role in experiencing negative emotion and processing aversive events such as pain (Levin, Lazrove, van der Kolk, 1999; Mollet & Harrison, 2006). Chemtob, Tolin, van der Kolk, Pittman (2000) support this lateralization hypothesis in PTSD; suggesting high levels of emotional arousal is linked to over-activation of the right hemisphere, interfering with adequate cognitive processing which enhances threat expectancies (Mazzola et al. 2009). Increased left hemisphere activation correlates to the availability of more coping resources and more adaptive reorganisation of the traumatic experience (van der Kolk, 1994).

Figure 4 highlights left and right brain activity.

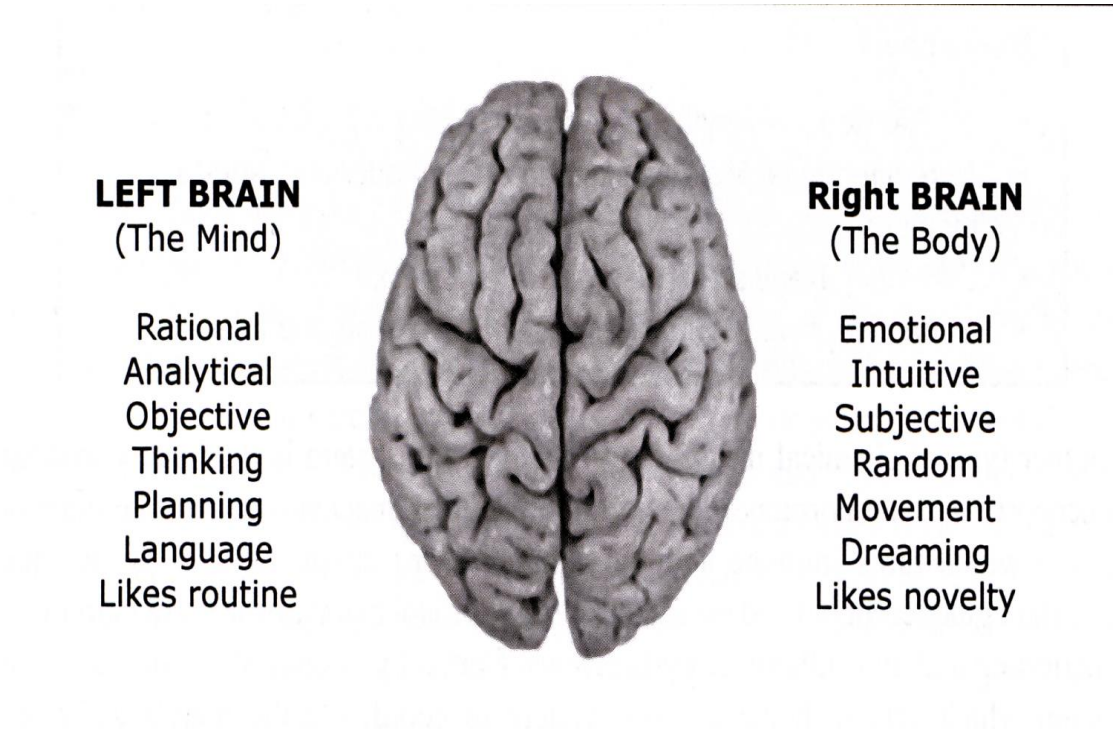


Figure 4: Left and right brain functions (from Grant, 2011, p. 37).

EMDR may also correct neurological abnormalities by stimulating both hemispheres promoting integration of the hemispheric functioning and normalization of brain activity (Levin et al. 1999; van der Kolk, 1996). Mazzola et al. (2009) cite a paper presented at the annual meeting of the EMDR International Association (in 1994) that used electroencephalography after EMDR to demonstrate that cerebral hemispheres were more synchronised.

This enhanced interhemispheric communication and cortical integration of traumatic memories leads to decreased negative emotional arousal with concomitant reduction in hypervigilance and decreased pain perception (Bergman, 1998; Levin et al. 1999; Ray & Zbik, 2001; van der Kolk, 1994). Bergman (1998) adds that EMDR increases activation of the anterior cingulate cortex and left prefrontal area enabling higher

brain functions to override input from limbic structures. Limbic down-regulation reduces sensitivity to pain.

EMDR treatment of CP and PLP includes processing the associated disturbing affective responses and the memories of the pain related events (Shapiro, 1995; 2001; 2002b). Ray and Zbik (2001) state the AIP accommodates "kindling" explanations of CP and neuroplasticity, as discussed in LAPS (Rome & Rome, 2000).

Neurobiological mechanisms: plasticity

Neuroplasticity in the somatosensory and motor systems are seen in neuropathic and musculoskeletal pain. In individuals with low back pain and fibromyalgia the amount of reorganisation increases with chronicity; in PLP and other neuropathic pain conditions plasticity is correlated with the amount of pain (Flor, 2003).

These central alterations may be seen as pain memories influencing the processing of painful and non-painful input (Katz & Melzack, 1990). Based on changes in the brain, these memories are not open to conscious awareness but lead to behavioural and perceptual changes outside of the individual's control (Flor, 2003). Pain memories are also implicated in PLP, as pain (prior to amputation) is a more important predictor of later PLP, than acute pain at time of amputation (Flor, 2003). Peripheral changes related to amputation may also contribute to enhanced cortical reorganization and PLP (Calford & Tweedale, 1991).

Cortical plasticity in CP can be modified by behavioural interventions that provide feedback to the brain areas that were altered by pain memories (Flor, 2003), such as habituation, sensitisation, operant and classical conditioning (Flor, Lutzenberger, Knost, Diesch, Birbaumer, 2002; Flor, Knost, Birbaumer, 1997; 2002). Learning affects pain behaviours, the subjective experience of pain, and the physiological processing of painful stimulation (Flor, 2003).

However, the difference between EMDR outcomes and CBT (cessation of pain versus decrease in pain (Ray & Zbik, 2001) is explained by neurobiological theories on the differences between memory reconsolidation and extinction (Suzuki, Josselyn, Frankland, Masushige, Silva, Kida, 2004). Treatments relying on extinction (such as exposure therapy) result in the formation of competing memories, rather than an alteration of the old ones. It is believed that EMDR's effectiveness is through reconsolidation which changes and restores the altered targeted memory itself (Suzuki et al. 2004).

Another theory of how EMDR (and CBT) may act is based on the homunculus (a sensory map of body parts in the brain) (see figure 5) and plasticity. The homunculus, present from birth is refined as we grow and do new things (Butler & Moseley, 2003). Suggesting a use-dependant brain, as the body parts used most are more largely represented in the homunculus. In PLP or CP the brain area related to the missing/ painful body part is "smudged", making that body part difficult to use or see (Butler & Moseley, 2003). Concentrating on phantom limb movement or performing a painful movement would activate the part of the homunculus related to

the missing/painful body part making it less “smudged” and leading to ultimately less pain (Butler & Moseley, 2003).

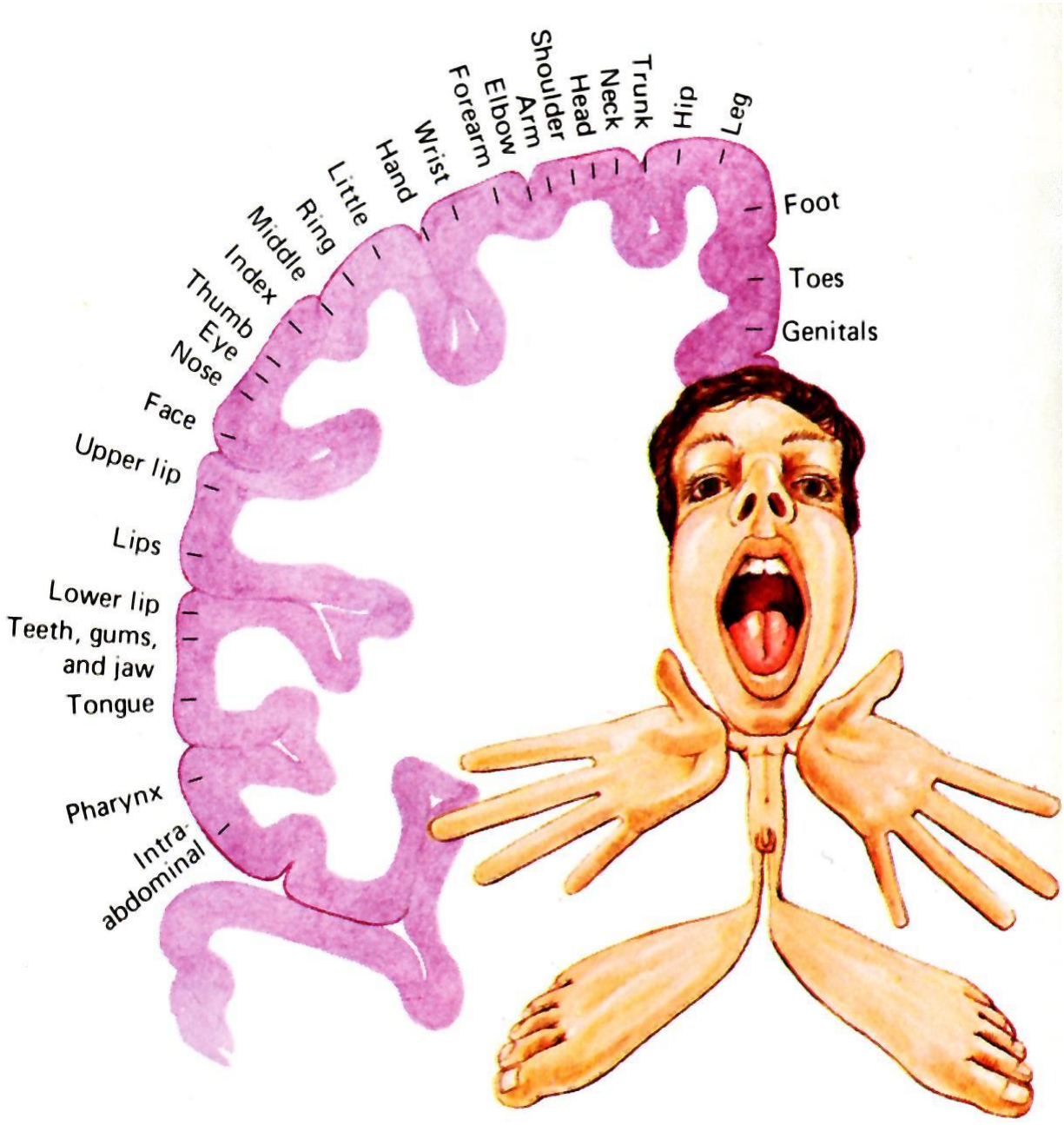


Figure 5: The homunculus. The sensory input from each body part proportioned according to the amount of cerebral cortex devoted to it (from Solomon, Schmidt, Adragna, 1990, p. 502).

The influence of affect and relaxation

Attention, hope, and/or support from a therapist may have contributed to pain reduction. However, this is unlikely as the subjects generally had long-standing CP and had not responded previously to pharmacological, psychological and complimentary treatments (de Roos et al. 2010).

Grant and Threlfo (2002) report alongside reduced pain levels, they saw reduced affective distress, and an increased perceived ability to cope with pain alongside less passive coping strategies (for example prayer) and maladaptive strategies (catastrophising). Learning is implicated and facilitated through Shapiro's (2001) AIP Model. In experiencing their pain differently, subjects make positive cognitive self-appraisals moving towards adaption from their own learning process (Grant & Threlfo, 2002).

Ray and Zbik (2001) suggest EMDR desensitizes emotional aspects of the pain experience. The involvement of the limbic system in pain perception/nociception is recognised (Butler & Moseley, 2003). Thus connections between traumatic memories and painful associations are separated with EMDR allowing individuals to experience their memories with less distress (Grant & Threlfo, 2002). If the emotional component of CP is altered it may change the way pain is "remembered", perceived and reproduced within the nervous system (central sensitization and such) (Grant & Threlfo, 2002; Mazzola et al. 2009) challenging cognitive appraisals and behavioural responses to influence treatment motivation and compliance (Getchel, Peng, Peters, Fuchs, Turk, 2007). An increasing awareness of the role of emotions in CP

suggests that psychological approaches that work directly through emotions may be very effective (Barber, 1997; Le Doux, 1996; Melzack, 1990; Ray & Zbik, 2001).

Although there is no formal relaxation in EMDR, it exerts a relaxation effect (Wilson, Silver, Covi, Foster, 1996) through bilateral stimulation (Mazzola et al. 2009). Relaxation allows the release of endogenous opiates which when combined with amygdala inhibition leads to the amelioration of symptoms and body sensations (Bergman, 1998; Grant & Threlfo, 2002). Friedberg (2004) also identified a stress reduction and relaxation effect in EMD.

Relaxation in itself has been shown to have some impact upon an individual's ability to control pain intensity through the initiated "relaxation response" (negating stress through (among others): decreased heart rate, decreased respiratory rate, lower blood pressure, decreasing muscle tension to reduce pain (Munden, Eggenberger, Goldberg, Howard, Mayer, Munson, 2003). Without regular use, improvements with relaxation are short-lived and do not increase the ability to control pain (Turk & Rudy, 1991; Turner, 1982). However pain reduction was seen at 2 year follow-up suggesting that the relaxation effect of EMDR did not contribute to pain reduction long-term, implicating other mechanisms (Grant & Threlfo, 2002).

Schneider et al. (2007) propose treatment is incomplete unless individuals gain new insights and an enhanced sense of self with the desire to help others in their pain. Wilensky (2006) also noted an increased positive sense of self, increased self-efficacy and self-determination with associated behavioural indicators of change following EMDR. Thus EMDR success rests not just on pain elimination (Schneider

et al. 2007). This growth is akin to outcomes in PCC (McMillan, 2004; Mearns & Thorne, 2000; 2007; Merry, 2002). Whether this is in part influenced by the number and length of EMDR sessions remains to be seen.

Lacking from the primary research was an exploration of fear in respect to negative cognitions and maladaptive behaviour. In my pain journey (the first two years in particular) fear was paramount, making my world shrink. Fear was also the crux in attempting to broaden my world and I believe I would never have been able to do that without someone who understood, empathised, and accepted me and my experiencing without judgement. I believe therefore that PCC would (theoretically) be effective in CP, whether as a single approach or part of a pluralist approach, research needs to determine.

The treatment of CP is therefore as complex as the mechanisms involved in its maintenance and perpetuation, and uniquely individual. Therefore a variety of approaches may be more appropriate than a one-size-fits-all approach to CP treatment. Although the results presented are promising, further RCTs, case studies, and case series are warranted because these approaches may contribute to establishing the efficacy of trauma-focused approaches to PLP, traumatic CP and non-traumatic CP, and may help to elucidate the processes involved (de Roos et al. 2010).

Critical discussion of limitations

Having worked within CP as a nurse and also as a CP sufferer, I hold opinions, insight and knowledge concerning CP, which may have broadened and constrained

this research. Despite endeavouring to "bracket off" aspects of my personal/professional experience, it was impossible to be free from my beliefs and assumptions about the investigated phenomenon (Stubbs & Bozarth, 2006).

Etherington (2004, p. 42) notes that "it seems to be generally accepted now that the topics we choose to research often have some personal significance for the researcher, whether conscious or unconscious". I concede that investigating this area may have been an attempt to validate aspects of my own beliefs and experience (Malhotra-Bentz & Shapiro, 1998). Aware of my internal biases, I attempted to reach and maintain transparency, openness and honesty by following SR guidelines and a more positivist stance.

It has been suggested that a literature review may be less valuable when a field is immature (too few studies to yield data) (Ravetz, 1973). However SRs can highlight the absence of data and point up the fact that any understanding is based on limited empirical underpinnings-in itself an important contribution- and direct future research efforts (Petticrew & Roberts, 2006). Furthermore, even when a field is immature, it is important to cumulate prospectively rather than wait for some later date when "enough" evidence is accumulated, and consolidation can occur (Petticrew & Roberts, 2006). Cumulating data can help in the early identification of effective interventions (Petticrew & Roberts, 2006).

This SR has highlighted gaps in the literature of a newly emerging research area. There were a small number of studies of varying quality which may have negatively impacted upon the results. For instance, the subjects may not be representative of

the general CP population as the sample was heavily weighted toward female participants (47 women to 20 men), with a variety but not representative sample of CP conditions. Berkley (1997) demonstrated a sex difference in pain, with a greater prevalence in women to men. This is explained by the interaction of stress in pain and the role of oestrogen leading to increased cortisol production (Berkley, 1997).

The primary studies were conducted in Australia (Grant, 2000; Grant & Threlfo, 2002), America (Friedberg, 2004), Canada (Wilensky, 2006), Germany (Schneider et al. 2007; 2008), Netherlands (de Roos et al. 2010) and Argentina (Mazzola et al. 2009). Not all ethnicities are represented potentially impacting upon the results and the representativeness of the sample.

The variety of primary studies was also diverse making a synthesis of findings problematic. The primary studies were all flawed to lesser or greater extents, lacking internal and external validity. As Muir Gray (2001) notes, primary studies that have been shown to have design flaws demonstrate exaggerated beneficial effects of interventions (Muir Gray, 2001). Likewise, systematically reviewing many biased studies does not produce an unbiased summary (Petticrew & Roberts, 2006).

In non-experimental investigations (case study, case series) the confounding of results is likely as the experimenter is unable to control all variables (Coolican, 1990). None of the studies were controlled so it cannot be ruled out that the results may be due to placebo effect or the result of spontaneous recovery. The latter being unlikely since most individuals in the studies had described having their pain for a long time and had often received other treatments without success. However no controlled

studies were available thus there was no rationale to exclude uncontrolled studies (Petticrew & Roberts, 2006).

Using my own evaluation of methodological quality of studies, rather than a validated tool may potentially threaten the validity of the SR outcome (Cooper, 1998). However, my rationale as demonstrated in the tables of the individual studies (Tables 4-11, Appendix III) is explicit. I was unable to locate two papers (concerning headache) through interlibrary loans. Cooper (1998) questions to what lengths a synthesis researcher should go to retrieve these documents. Following his process with a time limitation I decided not to explore other methods of obtaining them. Incomplete reporting by primary researchers also compromises the validity of the synthesis (Cooper, 1998) as I discovered.

The many types of pain complaint and a small non-representative sample does not allow for more specific conclusions. This SR offers that SP EMDR is effective in reducing pain intensity in PLP; furthermore that PP EMDR is effective in reducing pain intensity in other CP states, based on 8 studies: 2 case studies, 4 case series, 2 uncontrolled clinical trials. In total 68 (excluding 2 duplicated) individuals received EMDR for a CP complaint. All studies used a pre-test post-test design and half provided follow-up data.

The duration of treatment was generally short enough to make it unlikely that other factors (e.g., change in patient and/or life circumstances) influenced the outcome of these studies. EMDR may have a positive and clinically relevant effect on pain

intensity in PLP individuals. It is too early to make claims for the effectiveness of EMDR (PP) in CP.

Suggestions for further research

Further research using large studies with high internal and external validity is indicated by this SR. Case studies, case series and RCT's are needed to more reliably assess EMDRs effectiveness in PLP and traumatic and non-traumatic CP. McLeod (2002) suggests case studies may establish whether particular interventions are associated with beneficial outcomes and states a sound methodology allows for statistical analysis of several case studies. Case studies do not allow generalization of results to other groups of individuals RCTs would (McLeod, 2002). Since CP is heterogeneous, specific inclusion/exclusion criteria would demonstrate the effectiveness of EMDR for specific CP sub-groups (with and without PTSD for instance).

The EMDR protocol with targets identified will also facilitate generalization of results (van Rood & de Roos, 2009). Which target should be selected first (pain or trauma) for optimal results is worthy of further investigation. Control groups or a placebo intervention (relaxation training) ensure the results are due to the intervention (van Rood & de Roos, 2009). Longer follow-up will enable it to be seen if the positive results are maintained, and larger more representative samples will facilitate generalizability.

The integration of EMDR into current practice modalities (CBT, PCC for example) will also be an interesting area for further research as I am currently questioning if there

is a need for EMDR and PCC to be used in CP counselling individually or in a more integrative/pluralist approach. I wonder if elements of this dissertation (fear, memory, neuroplasticity) may also inform practice in those with difficulties such as panic and anger, as I can see theoretical links.

Much of the research into PTSD (with and without CP) and EMDR concerns (American) veterans. The evidence base, as I discovered, is limited in civilian use. Potential differences between the efficacy of EMDR in veteran and civilian populations may increase knowledge about memory, fear and neuroplasticity with huge potential to widen the audience for EMDR with perhaps protocol modifications to suit specific scenarios, such as PP EMDR.

CONCLUSION

The success of EMDR in this SR over a short number of sessions is important to individuals for their own empowerment and personal recovery; for therapists to be able to facilitate a real and lasting amelioration of suffering; and for policy-makers given the high incidence of traumatic and non-traumatic pain and the economic burden to health and social care services (Schneider et al. 2008). The cautiously positive results of this SR, that EMDR has the potential to be a useful treatment of CP and PLP; will hopefully lead to further research and more substantive results.

This dissertation has facilitated personal and professional knowledge of the mechanisms involved in PTSD and CP with a greater understanding of my own CP and in PTSD and CP of the individuals I have/am working with as a PCC. This

dissertation has also allowed me to explore ways of working outside of a uni-model approach and how I may integrate that into future person-centred practice.

I would like to specialise in counselling those with CP and PTSD (either co-occurring or individually). As such I intend to undertake a PhD to further investigate the use of EMDR in non-traumatic CP. I also plan to train in EMDR to enhance my practice in CP and PTSD. It feels exciting to be part of a new and growing theoretical and practical application of therapy, and I want to be actively playing a part in that evolution. Not only to increase my knowledge and understanding but to increase awareness of CP and PTSD to other counsellors/therapists to help reduce individual suffering.

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APPENDICES

Appendix I: Co-occurrence of PTSD and CP followed by references

Appendix II: Results of literature searches

Appendix III: Tables to show critical analysis of each primary study used within the research synthesis

APPENDIX I: Co-occurrence of PTSD and CP

Of those with diagnosed PTSD, 80% of combat veterans (Beckham, Crawford, Feldman, Kirby, Hertzberg, Davidson, et al. 1997) and up to 75% of individuals following a motor vehicle accident (Hickling, Blanchard, Silverman, Schwarz, 1992) also reported CP. Further, Geisser, Roth, Bachman, Eckert (1996) found significantly higher self-reported pain in individuals with high PTSD symptom load. Likewise, clinically relevant PTSD symptom severity was found in over 50% of individuals with CP conditions such as whiplash injury and Fibromyalgia (Sherman, Turk, Okifuji, 2000).

Further, recent research indicates that individuals suffering from CP with comorbid PTSD symptoms experience more intense pain, affective distress (Geisser, Roth, Bachman & Eckert, 1996) and higher disability (Duckworth & Iezzi, 2005; Palyo & Beck, 2005) than those without PTSD symptoms. Findings from community-dwelling adults (*n*=85,088) from 17 countries indicate that those with back or neck pain are two to three times more likely to have had in the past 12 months panic disorder, agoraphobia, or social anxiety disorder, and almost three times more likely to have had generalized anxiety disorder or PTSD (Demyttenaere, Bruffaerts, Lee et al. 2007).

Upwards of 30% of individuals seeking out-patient treatment for PTSD from community and mental health clinics report CP (Asmundson, Coons, Taylor & Katz, 2002; Otis, Keane & Kerns, 2003). Sareen, Cox, Clara, Asmundson (2005) in a National co-morbidity study in the United States, found that those with PTSD were

2.5 times more likely to have had a bone or joint condition (arthritis, rheumatisms, among others) in the past year. They conclude that CP and anxiety disorders, particularly PTSD, panic disorder, generalized anxiety disorder, social anxiety disorder, frequently co-occur (Sareen et al. 2005).

However despite the well-documented co-occurrence of the disorders (Asmundson & Katz, 2009) the nature of the relation between the conditions and potential mechanisms by which they are linked are still poorly understood (Asmundson et al. 2002; Asmundson & Katz, 2009).

Potential mechanisms of co-occurrence

Pain symptoms and CP are prevalent in individuals with PTSD, and PTSD symptoms are common in individuals with CP; particularly in those with higher pain severity, more interference in daily living, and higher negative affect. There are several possible relations scenarios: 1) PTSD and CP co-occur but are unrelated; 2) one causes the other; 3) each influences the other in some way through exacerbation of symptoms; 4) some other factor causes both (a genetic predisposition for instance). The evidence reviewed does not support points 1 and 2, and Asmundson et al. (2002) suggest that PTSD and CP co-occurrence literature reveals the suggestion of several mechanisms through which they are closely linked and influence each other as discussed below.

Shared Vulnerability

There may be predisposing factors facilitating the development of one or both conditions. Individual difference factors, possibly genetically influenced, predispose

individuals to develop PTSD and chronic musculo-skeletal pain (CMSKP) when exposed to certain environmental conditions. Specifically, the model suggests that the interaction of a psychological vulnerability for feelings of loss of control and anxiety, a lowered physiological threshold for alarm reactions (i.e. activation of fight/flight/freeze) to stressors, and instigating stressful events (e.g. traumatic incident, injury) all influence negative emotional responses, their consequences (physiological, behavioural and cognitive effects), and explain the development of PTSD, CMSKP and their co-occurrence (Asmundson et al. 2002; Otis, Keane, Kerns, 2003).

Co-occurring PTSD and CMSKP are most likely to develop in cases where vulnerable individuals are exposed to an event that is both traumatic and painful, wherein reminders of the trauma and sensations of pain can serve as triggers for further alarm reactions. The latter is consistent with postulates of the mutual maintenance model and further illustrates how predisposing factors can contribute to maintenance of these conditions (Asmundson & Katz, 2009). Asmundson and Hadjistavropoulos (2007) have suggested that symptoms of anxiety and pain may interact to promote clinically significant distress or impairment in persons with other anxiety disorders

Such potential predisposing factors include constructs of trait negative affectivity, harm avoidance and most promisingly, anxiety sensitivity (AS). AS denotes a tendency to become fearful, and more specifically, fearful of anxiety symptoms based on the belief that they may have harmful consequences (Reiss & McNally, 1985; Taylor, 1999). Taylor (1999) suggests three dimensions of AS: fear of publicly

observable anxiety reactions, fear of cognitive dyscontrol, and fear of somatic sensations. AS has been shown to be elevated in individuals with PTSD (Taylor, Fedoroff, Koch, Thordarson, Fecteau, Nicki, 2001; Taylor, Koch, McNally, 1992). Also in some (Kuch, Cox, Evans, Shulman, 1994), but not all samples of individuals with CP (Asmundson, Norton, 1995; Plehn, Peterson, Williams, 1998).

The severity of AS is positively correlated with severity of PTSD symptoms (Fedoroff, Taylor, Asmundson, Koch, 2000). Asmundson et al. (2002) suggest that AS amplifies the intensity of emotional reaction, particularly fear and anxiety and that elevated AS may be a predisposing factor for PTSD and CMSKP (Asmundson, Norton, Norton, 1999; Asmundson, 1999), i.e. AS predates the development of CP or PTSD and perpetuates each condition through highly intense emotional reaction to either a stressor or pain, fear, anxiety, and associated avoidance responses which increases the likelihood that pain will be maintained over time (Asmundson, 1999; Asmundson, Taylor, 1996; Muris, Vlacyen, Meesters, Vertongen, 2001; Vlacyen, Linton, 2000).

When the traumatic stressor and pain-precipitating event are the same or occur in close temporal proximity, AS may increase vulnerability for development of both conditions (Asmundson et al. 2002). It has yet to be established that elevated AS recedes the development of PTSD and CMS P; thus it remains a possibility that AS becomes elevated as a consequence of PTSD and CMSKP and thereafter serves to maintain symptoms (Asmundson, Norton, Norton, 1999). Longitudinal studies in PTSD and across the spectrum of anxiety disorders co-occurring with CP are needed to assess these possibilities (Asmunson & Katz, 2009).

Some evidence suggests a genetic basis for AS (Jang, Stein, Taylor, Livesley, 1999; Stein, Jang, Livesley, 1999); and PTSD (Stein, Jang, Taylor, Vernon, Livesley, 2002) and pain (Backonja, 1997; McCarson & Enna, 1999). There may be genetic factors common to all three conditions. Interestingly pain and anxiety disorders have highlighted genetically based dysregulation in serotinergic or GABA-ergic systems (Dessein, Shipton, Cloetey 1997; Kjorsvik, Storkson, Tjolsen, Hole, 1997; McCarson & Enna, 1999).

Thus it appears plausible that AS represents a bridge or shared vulnerability between PTSD and CP.

Mutual maintenance

Sharp and Harvey (2001) posit that some cognitive, affective and behavioural components of CP maintain or exacerbate symptoms associated with PTSD. Likewise, some components of PTSD (physiological, affective and behavioural) maintain or exacerbate symptoms associated with CP. Essentially, CP serves as a persistent reminder of the trauma and conversely that arousal triggered by the reminder promotes avoidance of pain-related situations. Over time, physical deconditioning makes the experience of pain more likely trapping the individual in a vicious cycle whereby the symptoms of PTSD and CMSKP interact to produce self-perpetuating distress and functional disability. Sharp and Harvey (2001) propose seven mechanisms through which mutual maintenance occurs: 1) attentional and reasoning biases; 2) AS; 3) reminders of the trauma; 4) avoidance; 5) depression and reduced activity levels; 6) anxiety and pain perception; 7) cognitive demand from symptoms that limits the use of adaptive strategies (e.g. catastrophising).

Gil, Caspi, Ben-Ari, Koren, and Klein (2005) highlighted the role of reminders of the trauma in triggering arousal and other PTSD symptoms, and of CP conversely being a constant reminder of the traumatic event. Increased arousal enhanced the pain perception through changes in pain transduction pathways in the brain. These mechanisms were tested by Asmundson et al. (2002); Beck, Gudmundsdottir, Shipherd (2003); Duckworth and Iezzi (2005); Palyo and Beck (2005); Poundja, Fikretoglu, Brunet (2006) suggesting that these factors do impact on the development of CP either on a direct or indirect pathway (Jenewein, Wittmann, Moergeli, Creutzig, Schnyder 2009).

This model has been criticised, by Asmundson et al. (2002) for confusing the concepts of shared vulnerability and mutual maintenance, over-simplifying point 3 (reminders of the trauma) and for suggesting that feedback loops are more likely to be uni-directional whereas Asmundson et al. (2002) suggest they are bi-directional on occasion. Furthermore, most of the aforementioned studies used cross-sectional designs thereby limiting the inferences on the nature of the relation between PTSD symptoms and CP symptoms (i.e. mutual influence, influence of pain on PTSD or vice versa). Jenewein et al. (2009) in a 12 month longitudinal study of 335 accident survivors confirmed the mutual maintenance model (Sharp & Harvey, 2001) in the early aftermath of the accident only. From 6 months post accident onwards, higher PTSD symptom levels were associated with increased pain intensity but not vice versa.

Further research is required to determine if, for any given individual, PTSD or CP came first, as this may enable preventative treatments to be administered to the

individual (Asmundson et al. 2002). Research also needs to explore further the extent to which PTSD and CP are related (Asmundson et al. 2002). Trauma may induce changes in biological substrates altering arousal mechanisms and pain transduction pathways in the brain (Jenewein et al. 2009). Studies of co-occurring PTSD and CP are potentially confounded because a traumatic event involving personal physical injury (e.g. road traffic accident, work related or combat injury often precipitates both pain and PTSD reactions.

Hitherto a clear link between the co-occurrence of CP and PTSD has been established with two models proposed explaining underlying mechanisms.

Selective attention to threat

There is considerable evidence indicating that individuals with various forms of psychopathology and general medical conditions selectively attend to threat related stimuli representative of the core concerns of their specific disorder; they direct attention toward objects or situations that they fear. This increases state anxiety and has potential to make one vulnerable for emotional disorders (Matthews, 2002; Williams, Matthews, MacLeod, 1996). An underlying assumption of attentional bias research in individuals with CP is that these individuals are fearful of pain, view it as a threat, and thus, selectively direct attention to pain-related stimuli (Asmundson & Katz, 2009). However, investigators may not have correctly identified the specific object/s or situations that are feared (Morley & Eccleston, 2004). Pain related stimuli may not be the only object of fear for many individuals with CP as several studies indicate that prior trauma related stimuli may be the most relevant object of fear (Asmundson & Katz, 2009).

Lower threshold for alarm

Pain and anxiety are both associated with autonomic nervous system (ANS) physiological arousal (accelerated heart rate, elevated blood pressure, increased respiration rate, decreased gastrointestinal activity, increased muscular tension, increased blood flow to skeletal muscle) and the release of neural and hormonal substances associated with the "stress response" (Kaptein & Weinman, 2004). These physiological mechanisms of arousal serve a protective function, fight/flight. Prolonged physiological arousal, whether initiated by pain or anxiety, act as stressors; contributing to the perception of threat and uncontrollability, having detrimental effects on various bodily systems (Kaptein & Weinman, 2004).

ANS arousal is thought to contribute to the symptoms of PTSD and CP (Keane, Kolb, Kaloupek, 1998). Rainville, Bao, Chretien (2005) demonstrated pain-related emotion impacts in ANS responsivity in individuals without CP or PTSD. Other literature suggests dysregulation of the endogenous opioid system may blunt pain perception, reducing pain avoidance behaviour and increasing emotional numbing associated with CP and PTSD (Geuze, Westenberg, Jochims et al. 2007). However this remains to be evaluated in direct comparisons between those with PTSD; CP; both PTSD and CP; and clinical control participants.

The mutual maintenance and shared vulnerability models provide a framework to guide further research, perhaps exploring CP co-occurring alongside other anxiety disorders other than PTSD.

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APPENDIX II: Results of the literature searches

1. **EMDR and chronic pain**
2. **EMDR and phantom limb pain**
3. **EMDR and somtof***
4. **EMDR and headache**
5. **Email from Chester University library RE: British Library unable to locate a paper**

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Searching: **CINAHL Plus with Full Text,** Show all | Choose Databases

EMDR in Select a Field (optional) Search

AND chronic pain in Select a Field (optional) Clear

AND in Select a Field (optional) Add Row

Basic Search | Advanced Search Visual Search | Search History

Note: The number of available results reflects the removal of duplicates.

1. **EMDR** in the Treatment of **Chronic Pain**.

By: Grant, Mark; Threlfo, Catherine. Journal of Clinical Psychology. Dec2002, Vol. 58 Issue 12, p1505-1520. 16p. 4 Graphs.

Subjects: EYE movement desensitization & reprocessing; **CHRONIC pain** -- Treatment

Database: Psychology and Behavioral Sciences Collection

PDF Full Text (2.5MB) | Link to Full Text Check 360 Link for Full Text

2. EMDR in the Treatment of Chronic Phantom Limb Pain.

(includes abstract); Schneider J; Hofmann A; Rost C; Shapiro F; **Pain** Medicine, 2008 Jan-Feb; 9 (1): 76-82 (journal article - case study, research) ISSN: 1526-2375 PMID: 18254770

Subjects: Desensitization, Psychologic; Eye Movements; Phantom Limb; Adult: 19-44 years; Aged: 65+ years; Middle Aged: 45-64 years; Female; Male

Database: CINAHL Plus with Full Text

Link to Full Text Check 360 Link for Full Text

3. **EMDR** and the Role of the Clinician in Psychotherapy Evaluation: Towards a More Comprehensive Integration of Science and Practice.

By: Shapiro, Francine. Journal of Clinical Psychology. Dec2002, Vol. 58 Issue 12, p1453-1463. 11p.

Subjects: EYE movement desensitization & reprocessing; POST-traumatic stress disorder -- Treatment; PHOBIAS -- Treatment; **CHRONIC pain** -- Treatment

Database: Psychology and Behavioral Sciences Collection

PDF Full Text (1.8MB) | Link to Full Text Check 360 Link for Full Text

4. Treatment of **chronic** phantom limb **pain** using a trauma-focused psychological approach.

(**eng**)Abstract Available By: de Roos C, Veenstra AC, de Jongh A, den Hollander-Gijsman M, van der Wee NJ, Zitman FG, van Rood YR, **Pain** Research & Management: The Journal Of The Canadian **Pain** Society = Journal De La Société Canadienne Pour Le Traitement De La Douleur [**Pain** Res Manag], ISSN: 1203-6765, 2010 Mar-Apr; Vol. 15 (2), pp. 65-71; PMID: 20458374

Subjects: Amputation, Traumatic psychology; Behavior Therapy methods; Pain etiology; Pain psychology; Phantom Limb complications; Adult: 19-44 years; Aged: 65+ years; Middle Aged: 45-64 years; All Adult: 19+ years; Female; Male

Database: MEDLINE

Link to Full Text Check 360 Link for Full Text

5. EMDR in the treatment of chronic pain.

Mazzola, Alexandra Calcagno, Marìa Luján Goicochea, Marìa Teresa Pueyrredòn, Honorio Leston, Jorge Salvat, Fernando ; Journal of **EMDR** Practice and Research, Vol 3(2), 2009. pp. 66-79. [Journal Article]

Subjects: Chronic Pain; Eye Movement Desensitization Therapy; Adulthood (18 yrs & older); Young Adulthood (18-29 yrs); Thirties (30-39 yrs); Middle Age (40-64 yrs); Aged (65 yrs & older); Male; Female

Database: PsycINFO

Link to Full Text Check 360 Link for Full Text

6. **EMDR**: a new treatment for trauma and **chronic pain**.

(**eng**)Abstract Available By: Grant M, Complementary Therapies In Nursing & Midwifery [Complement Ther Nurs Midwifery], ISSN: 1353-6117, 2000 May; Vol. 6 (2), pp. 91-4; PMID: 10844748

Subjects: Desensitization, Psychologic methods; Eye Movements; Pain psychology; Pain Management; Wounds and Injuries psychology; Wounds and Injuries therapy; Adult: 19-44 years; All Adult: 19+ years; Female

Database: MEDLINE

Link to Full Text Check 360 Link for Full Text

7. A single session of hypnosis and eye movement desensitisation and reprocessing (EMDR) in the treatment of chronic pain.

Ray, Patricia Page, Andrew C. ; Australian Journal of Clinical & Experimental Hypnosis, Vol 30(2), Nov, 2002. pp. 170-178. [Journal Article]

Subjects: Chronic Pain; Eye Movement Desensitization Therapy; Hypnotherapy; Multimodal Treatment Approach; Adulthood (18 yrs & older); Male; Female

Database: PsycINFO

Link to Full Text Check 360 Link for Full Text

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Searching: **CINAHL Plus with Full Text,** Show all | Choose Databases

phantom limb pain in Select a Field (optional) Search

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Basic Search | Advanced Search | Visual Search | Search History

Note: The number of available results reflects the removal of duplicates.

1. **EMDR in the Treatment of Chronic Phantom Limb Pain.**

(includes abstract); Schneider J; Hofmann A; Rost C; Shapiro F; **Pain** Medicine, 2008 Jan-Feb; 9 (1): 76-82 (journal article - case study, research) ISSN: 1526-2375 PMID: 18254770

Subjects: Desensitization, Psychologic; Eye Movements; Phantom Limb; Adult: 19-44 years; Aged: 65+ years; Middle Aged: 45-64 years; Female; Male

Database: CINAHL Plus with Full Text

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2. **Treatment of chronic phantom limb pain using a trauma-focused psychological approach.**

(**eng**)Abstract Available By: de Roos C, Veenstra AC, de Jongh A, den Hollander-Gijsman M, van der Wee NJ, Zitman FG, van Rood YR, **Pain** Research & Management: The Journal Of The Canadian **Pain** Society = Journal De La Société Canadienne Pour Le Traitement De La Douleur [**Pain** Res Manag], ISSN: 1203-6765, 2010 Mar-Apr; Vol. 15 (2), pp. 65-71; PMID: 20458374

Subjects: Amputation, Traumatic psychology; Behavior Therapy methods; Pain etiology; Pain psychology; Phantom Limb complications; Adult: 19-44 years; Aged: 65+ years; Middle Aged: 45-64 years; All Adult: 19+ years; Female; Male

Database: MEDLINE

Link to Full Text Check 360 Link for Full Text

3. **Eye movement desensitization and reprocessing (EMDR) in the treatment of war veterans.**

By: Silver, Steven M.; Rogers, Susan; Russell, Mark. Journal of Clinical Psychology. Aug2008, Vol. 64 Issue 8, p947-957. 11p. DOI: 10.1002/jclp.20510.

Subjects: EYE movement desensitization & reprocessing; VETERANS; TRAUMATISM -- Treatment; POST-traumatic stress disorder; PSYCHOTHERAPY; ANXIETY; MENTAL depression; ANGER; **PAIN**; META-analysis

Database: Psychology and Behavioral Sciences Collection

PDF Full Text (114KB) | Link to Full Text Check 360 Link for Full Text

4. **EMDR and phantom limb pain: Theoretical implications, case study, and treatment guidelines.**

Schneider, Jens Hofmann, Arne Rost, Christine Shapiro, Francine ; Journal of **EMDR** Practice and Research, Vol 1(1), 2007. pp. 31-45. [Journal Article]

Subjects: Eye Movement Desensitization Therapy; Pain; Phantom Limbs; Treatment Guidelines; Adulthood (18 yrs & older); Thirties (30-39 yrs); Male

Database: PsycINFO

Link to Full Text Check 360 Link for Full Text

5. **Treating traumatic amputation-related phantom limb pain: A case study utilizing eye movement desensitization and reprocessing within the armed services.**

Russell, Mark C. ; Clinical Case Studies, Vol 7(2), Apr, 2008. pp. 136-153. [Journal Article]

Subjects: Amputation; Eye Movement Desensitization Therapy; Pain; Phantom Limbs; Posttraumatic Stress Disorder; Adulthood (18 yrs & older); Young Adulthood (18-29 yrs); Male

Database: PsycINFO

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Searching: **CINAHL Plus with Full Text,** Show all | Choose Databases

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AND in Select a Field (optional) Add Row

Basic Search | Advanced Search | Visual Search | Search History

Note: The number of available results reflects the removal of duplicates.

1. **Eye movement desensitization and reprocessing in the psychological treatment of trauma-based psychogenic non-epileptic seizures.**

By: Kelley, Susan D. M.; Benbadis, Selim. Clinical Psychology & Psychotherapy. Mar/Apr2007, Vol. 14 Issue 2, p135-144. 10p. DOI: 10.1002/cpp.525.

Subjects: EYE movement desensitization & reprocessing; SPASMS; POST-traumatic stress disorder; DISSOCIATION (Psychology); **SOMATOFORM** disorders

Database: Psychology and Behavioral Sciences Collection

PDF Full Text (114KB) | Link to Full Text | Check 360 Link for Full Text

2. On treatment with eye movement desensitization and reprocessing of chronic post-traumatic stress disorder in public transportation workers--a randomized controlled trial.

(eng)Abstract Available By: Högberg G, Pagani M, Sundin O, Soares J, Aberg-Wistedt A, Tärnell B, Hällström T, Nordic Journal Of Psychiatry [Nord J Psychiatry], ISSN: 0803-9488, 2007; Vol. 61 (1), pp. 54-61; PMID: 17365790

Subjects: Desensitization, Psychologic; Eye Movements; Occupational Diseases epidemiology; Public Sector; Stress Disorders, Post-Traumatic therapy; Transportation; Adult: 19-44 years; All Adult: 19+ years; Female; Male

Database: MEDLINE

PDF Full Text Link to Full Text Check 360 Link for Full Text

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Searching: **CINAHL Plus with Full Text,** Show all | Choose Databases

EMDR in Select a Field (optional) Search

AND Headache in Select a Field (optional) Clear

AND in Select a Field (optional) Add Row

Basic Search | Advanced Search | Visual Search | Search History

Note: The number of available results reflects the removal of duplicates.

1. **EMDR treatment of migraine.**

Konuk, Emre Epözdemir, Hejan Atçeken, Şirin Haciömeroğlu Aydin, Yunus Emre Yurtsever, Asena ; Journal of **EMDR** Practice and Research, Vol 5(4), 2011. pp. 166-176. [Journal Article]

Subjects: Eye Movement Desensitization Therapy; Eye Movements; Migraine Headache; Posttraumatic Stress Disorder; Adulthood (18 yrs & older); Male; Female

Database: PsycINFO

Link to Full Text Check 360 Link for Full Text

2. **Phase 1 of integrated EMDR: An abortive treatment for migraine headaches.**

Marcus, Steven V. ; Journal of **EMDR** Practice and Research, Vol 2(1), 2008. pp. 15-25. [Journal Article]

Subjects: Eye Movement Desensitization Therapy; Medical Diagnosis; Migraine Headache; Adulthood (18 yrs & older); Young Adulthood (18-29 yrs); Thirties (30-39 yrs); Middle Age (40-64 yrs); Male; Female

Database: PsycINFO

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APPENDIX III: Tables 4-11, to show critical appraisal of primary studies included within the systematic review

1. **Grant, 2000**
2. **Grant and Threlfo, 2002**
3. **Friedberg, 2004**
4. **Wilensky, 2008**
5. **Schneider et al. 2007**
6. **Schneider et al. 2008**
7. **Mazzola et al, 2009**
8. **de Roos et al. 2010**

AUTHOR	AIM	STUDY	SUBJECTS	MAIN FINDINGS	COMMENTS
Grant, 2000	To demonstrate the Effectiveness of EMDR In the treatment of Chronic pain and how clients can be taught to self manage pain using EMDR components	Case study (Tanya excluded here as included with more detail in following paper) Sara -EMDR sessions for PTSD followed by EMDR sessions for CP Taught self-use of desensitization component of EMDR	Female 40yrs PTSD and CP (CP following accident)	After first session pain virtually disappeared (lasting over 12 hours) PSTD symptoms Considerably decreased After few weeks of self-directed desensitization and audio bilateral stimulation= whole pain free days and virtually sleeping through night	No tools used to measure pain pre/post intervention-subject report Sara's pain is traumatic in origin which may account for apparent amelioration of pain with EMDR a trauma-focused intervention Unknown number of sessions of EMDR used for PTSD or EMDR for CP- may have impacted on CP results Unknown what EMDR protocols were used Unknown what self-administered protocol was followed Unknown whether ethical approval was sought Unknown where subjects were recruited from No medium or long term follow up No inclusion/exclusion criteria No control group Study design appropriate for research question, however Poorly reported, lacks internal and external validity, rigour, and reliability. Low quality study.

AUTHOR	AIM	STUDY	SUBJECTS	MAIN FINDINGS	COMMENTS
Grant & Threlfo, 2002	To examine if EMDR (with self-directed EMDR at home) reduces emotional distress in CP and alleviates CP	Case series 9 weekly 1hour long EMDR sessions Measures of pain (short-form McGill Pain Questionnaire (SFMPQ) Cognitive & behavioural coping score (CSQ) taken at: pre-intervention After 5 sessions Post-intervention 2 month follow-up (F/up) 2 yr F/up Subjective Units of Distress Scale (SUD) & Validity of Cognition Scale (VoC) during EMDR treatment EMDR therapist experienced in CP	3 women with CP Jocelyn 27yrs, 4 year CP following vehicle accident Kylie 54yrs, 10 yr CP (hip) following broken leg Tanya 28yrs, 2 yr CP All described emotional distress Answered newspaper advertisement to Participate Inclusion criteria (accepting of medical diagnosis, pain levels tolerable, have CP for minimum 6months)	Pain reduced & Affective distress reduced Jocelyn: pain (pre =68 to 35 F/up) CSQ (pre=33 to15 F/up) Ability to control pain: (pre=3 to 4 F/up) Ability to ↓ pain (pre=3 to 5 F/up) Kylie: pain (pre= 72 to 30 F/up) CSQ (pre-45 to21 F/up) Ability to control pain: (pre= 0 to 6 F/up) Ability to ↓ pain: (pre= 0 to 6 F/up) Tanya: pain (pre= 45 to 32 F/up) CSQ (pre= 39 to 9) Ability to control pain: (pre=0 to 3.8 F/up) Ability to ↓ pain: (pre= 1 to 4 F/up) Only subject available at 2yr F/up: continued ↓ pain, ↑ mobility, ↑ ability to manage pain, ↓affective distress with original trauma	Clear EMDR protocol- delivered by experienced therapist (EMDR & CP) Nature of self-directing bilateral stimulation not given Results due to relaxation element of EMDR? Valid & reliable pain assessment tool (SFMPQ; Melzack, 1987) CSQ unknown if valid & reliable SUD validated and reliable in PTSD ?in CP VoC validated and reliable in PTSD ? in CP No tool to evaluate acceptance of medical diagnosis No definition of “tolerable limits” given for subjects pain No listing of specific questions (other than 1) used by therapist to facilitate cognitive associations Subjects all Caucasian women- subject bias. Good descriptive data No variety in ages, are results due to young age? Small sample size- not representative No control group- generalisability, reliability of results 2 subjects lost at 2yr F/up Study design appropriate for research question. Well written, controls attempted for internal validity. Lacks external validity. Intermediate quality study

AUTHOR	AIM	STUDY	SUBJECTS	MAIN FINDINGS	COMMENTS
Friedberg, 2004	Use of self-administered EMD in the treatment of pain, fatigue, and psychological symptoms in Fibromyalgia patients	Pilot study 2 (1 hourly) Sessions weekly Home EMD = 2x10min daily throughout study Session 1: pain intensity & location, functional Limitations, psychodiagnostic interview (based Upon DSM-IV) Pre-EMD intervention, end session 2 and 3mth F/up: SUD, Fibromyalgia Impac Scale (FIQ) (function, pain, stiffness, fatigue, sleep, depression, anxiety, well-being) fatigue Scale (FS) Beck Anxiety Inventory (BAI) Thermal biofeedback (indirect measure of blood flow indicating relaxation) EMD journal (home-assigned EMD session details, pre- & Post- ratings for pain, stress, fatigue)	6 Caucasian females Recruited from local Fibromyalgia and Chronic Fatigue Syndrome support Group. Aged 27-53yrs (mean43.2) Illness duration 4-20yrs (mean10.2yr) All subjects at 3mth F/up	Co-morbid psychiatric Disorders found in all subjects: 3=generalised anxiety; 3= dysthymia; 1=panic disorder During EMD biofeedback (mean hand temperature) ↑(average 6.6°F session1, average 4.1°F session2) indicating relaxation SUD ratings of pain ↓ (session1= 17.1%, session 2= 18.5%) All other measures showed improvements. 4 subjects identified as treatment responders (25% or more improvement from baseline to F/up In at least 2 out of 4 measures) 3mth F/up- 5 able to perform self-EMD no negative side effects, 1 reported ↑anxiety levels. Told to discontinue EMD- no further effects	EMD advocated over EMDR as in author's experience, EMDR may potentially trigger disturbing recollections Author posits EMD comparable to EMDR in effectiveness- ? evidence EMD protocol based on author's book- ? validated/reliable in CP Clear EMD protocol Experienced EMD therapist (qualifications and when attained stated) Validated tools- FIQ, FS, BAI ? SUD validated in Fibromyalgia No tool for pain location and severity No control group-generalisability difficulties Small sample size (representativeness affected) – Pilot study Good descriptive data for subjects Subject bias- all female Age bias- young subjects Future research- comparing EMD to EMDR in CP EMD successful at inducing relaxation, potential use as adjunct to CBT or PCC for various anxiety disorders-future research potential Study design appropriate to answer research question. Well written, attempts made to maintain internal validity lacks external validity. Intermediate quality study.

AUTHOR	AIM	STUDY	SUBECTS	MAIN FINDINGS	COMMENTS
Wilensky, 2006	To investigate the effectiveness of EMDR in PLP	5-Case Series Standard EMDR Protocol Pre-intervention: Impact of Event Scale-Revised (IES-R) Pain & Disability Index (PDI) Trauma Symptom Inventory (TSI) Beck Depression Inventory (BDI) Beck Depression Inventory 2nd edition (BDI-II) SUD VoC Author approached rehab unit, invited to work with psychology dept members watching behind glass – Identified 3 subjects Word of mouth= presentation of 2 subjects (Dave & Edith)	Al – 39yrs PLP foot 3months, EMDR sessions=5 Childhood physical trauma to foot (now amputated), childhood physical abuse, substance abuse, previous suicide attempt. Traumatic accident leading to amputation (fear of stairs & elevators since accident) Seen in rehab unit Bert- 25yrs PLP foot 2mths, EMDR sessions=3 Childhood physical abuse, raised by grandparents after mother left Traumatic accident leading to amputation Seen in rehab unit Chuck- 43yrs PLP arm 3yrs EMDR sessions=9 Traumatic accident leading to amputation Previous near death experience when electrocuted. Anything resembling electricity = pain trigger. Pain intensity led to suicidal ideation and voluntary hospitalisation Seen in rehab unit Dave-55yrs PLP finger 5mths EMDR sessions=8 Father misused alcohol & physically abused child Dave. Brother=traumatic death. Dave =drug (heroin & alcohol abuse. Multiple hand injuries before traumatic accident leading to amputation. Panic attacks Seen in author's clinical office Edith-66yrs PLP leg 1week EMDR sessions=3 (+2 with different therapist post discharge) Difficult adolescence. Amputation due to cancer Seen in rehab unit	Al- Pre-intervention: pain=10/10 IES-R=65 PDI= 45 Post-intervention: Pain=0 IES-R=25 PDI=14 Able to use stairs & elevators Bert- Pre-intervention: pain=7 IES=67 PDI= ? drinking 6 beers nightly Post-intervention: Wearing prosthesis, not done so previously, exercising at gym, driving & ↓alcohol consumption Pain=1 IES=35 PDI=? Happy with level of functioning chose to leave study Chuck- Pre-intervention: pain=8 BDI=34 PDI= 49 Post-intervention: Pain=1 BDI=11 PDI=22 Happy with level of functioning chose to leave study Dave- Pre-intervention: pain=5 IES=55 TSI= 6 scales >65 Post-intervention: Pain=1 IES=5 TSI=all<65 F/up 1yr+ treatment gains maintained Edith- Pre-intervention: pain=8-9 Post-intervention pain=0 No other measures given F/up 3yrs- no PLP (other occasional Pain noted	Valid & reliable measurement tools Not clear if all tools used on all subjects as not all tool measurements given for every subject Background stories to subjects & events concerning amputation Inconsistent reporting of some pain scores (Bert & Chuck) and age (Dave) different in a table, compared to case illustrations PLP at 1 week not classed as CP 4 men, 1 woman- subject bias. Various ages F/up 1yr for Dave (visit) F/up 3yrs Edith (letter) Author performed EMDR-standard protocol. Unknown level of experience/qualification. No standard number of sessions given in text but from discussion appears all but Chuck completed prescribed treatment Accident memories and earlier memories targeted by EMDR Pain ↓in all subjects, and all subjects saw increased positive sense of self Number of EMDR sessions correlated with amount of time since accident- sooner a client seen post amputation the quicker the remission? Study protocol lacks standardization of measurement tools, EMDR sessions & F/up. Poorly written with numerical discrepancies. Lacks internal and external validity. Low quality study.

AUTHOR	AIM	STUDY	SUBJECTS	MAIN FINDINGS	COMMENTS
Schneider, Hofmann, Rost, Shapiro, 2007	EMDR and Adaptive Information Processing (AIP) model in treatment of Phantom Limb Pain (PLP)	Case Study 9 sessions of EMDR (1 assessment) Standard EMDR protocol over 2 inpatient stay and 3 outpatient visits plus telephone contact. Structured clinical interview for DSM-IV. Criteria met for PTSD & major depressive disorder, Pre- intervention: Pain levels assessed (Visual Analogue Scale), Impact of Events Scale (IES), Beck Depression Scale (BDI), VoC, SUD. Type and dosage of medication F/up 1 year & 18months	Tom, 38 yrs, PTST and PLP (chronic pelvic pain same accident) for 3 years following motorcycle accident. Same subject from case series (below). Included here as omitted in case series Various pain & rehabilitation treatments & analgesic regimes After first hospital admission and completed F/up visits and telephone calls, Tom requested re-admission (↑ pain ↑ depression at night. Poor sleep). Tom had developed a night time routine looking at photographs of amputated leg and damaged body before bed (to desensitize himself)	Admit 1 (7 weekly EMDR sessions) Pre- VAS 10+, BDI 17, IES 60 Post- VAS 4.5, BDI 10, IES 18 Pre- morphine 600mg/day Non-steroidal anti-inflammatory (Metamizol) 2g/day, Gabapentin 1800mg/day Post- morphine 200mg/d, Metamizol 0mg, gabapentin 0mg Out-patient (3mth F/up): VAS 5, BDI 6, IES 18, morphine 100mg/d Admit 2 (3 weekly EMDR sessions) Pre- VAS 7.5, BDI 20, IES 45 Post- VAS 0.5, BDI 10, IES 8 Pre- morphine 300mg/d as required Post- morphine 100mg/d as required F/up 1 yr & 18mth: VAS psychometrics showed no trauma or depression, no PLP, pelvic pain 2-4. Medication rarely taken (morphine 100mg as required)	Standard EMDR protocol (8 steps) Specifically using AIP to target inadequately processed memories and emotions of PLP Relapse if missed reprocessing all triggers and associated memories PLP correlated with affective distress, supporting AIP theory Antidote imagery First author performed EMDR unknown level of experience/qualification status VoC & SUD not consistently or regularly displayed in text or table form No self-administered EMDR Pain ameliorated as by-product emerging during EMDR No apparent consent from subject (consent implied in admit 2) Admit 2 sessions conducted very close together, explaining some data omissions ethical approval from hospital in report Valid and reliable psychometric tools. Worked with Tom's changed self-concept Study design appropriate to answer research question. Well written. Lacks external validity, maintains internal validity. High quality study.

AUTHOR	AIM	STUDY	SUBJECTS	MAIN FINDINGS	COMMENTS
Schneider, Hofmann, Rost, Shapiro, 2008	EMDR in Treatment Of Phantom Limb Pain (PLP)	Case Series 3-15 sessions of EMDR S1=50mins of 15 sessions S2=90mins of 4 sessions S3= 90mins of 4 sessions S4= 90mins of 9 sessions Standard EMDR protocol Pre-post-intervention & F/up measures: Impact of events scale (intrusion & avoidance symptoms) (IES, 1-75) Beck Depression Inventory (BDI, 1-63) Visual Analogue Scale for pain Intensity (0-10) Type and dosage of medication	5 subjects with case illustration (Tom). Same subject focus of Case study therefore omitted from This study to avoid duplication. 4 subjects of unknown gender Aged 41-67 2 (half) left during study but not lost to F/up Hospital inpatients (x2) Hospital outpatients (x2) PLP 4-16 years	S1- pre- to post intervention Pain ↓(strong permanent to moderate every other day) IES ↓(46-15) BDI ↓(25-15) Medication: analgesia dose unchanged (morphine 100mg once daily) F/up (24months): pain levels unchanged, no psychometrics, medication unchanged S2- pre- to post intervention Pain ↓(10 - 0) IES ↓(67-18) BDI ↓(32-11) Medication: analgesia dose unchanged (Tilidin 150mg, gabapentin 1800mg, pregabalin 600mg, doxepin 50mg) Anti-anxiety/sedative medication stopped (Diazepam 40mg to 0mg) F/up (14mths): pain levels unchanged, no psychometrics, medication unchanged S3- pre- to post intervention Pain ↓(10-5) IES ↓(42-20) BDI ↓(12-10) Medication: analgesia dose reduced (morphine 180mg to 100mg) F/up (14mths): pain levels unchanged, no psychometrics, medication unchanged S4- pre- to post intervention Pain ↓(8 every 3rd week – 6 every 8th week) No psychometrics Medication: analgesia dose unchanged (Tilidin 150mg, Ibuprofen 600mg, (as required) Paracetamol 500mg). F/up (21mths): pain levels unchanged, no psychometrics, medication unchanged	Standard EMDR protocol (not CP) ? PTSD/traumatic memory implied Unknown which author or authors performed EMDR or their experience/ qualification status No self-administered EMDR ? why F/up intervals not standardised over subjects No psychometrics at F/up ?why (incomplete data collection) Not enough descriptive data on subjects and recruitment method (inclusion/exclusion)not explicit Subjects aged 41 (x2), 67, 59- more representative Small sample (4)-generalisability High non-completion of intervention rate (50% or 2 subjects)- generalisability No apparent consent from subjects or ethical approval from hospital in report Valid and reliable psychometric tools. Shows EMDR reduces affective & cognitive aspects to trauma and can reduce pain-sustained Study design appropriate to answer research question. Poorly written. Lacks external validity, attempts made to maintain internal validity. Low quality study.

AUTHOR	AIM	STUDY	SUBJECTS	MAIN FINDINGS	COMMENTS
Mazzola, Calcagno, Goicochea, Leston, Salvat, 2009	To investigate the use of EMDR in Chronic pain. 3 theories of mechanisms	Uncontrolled Study 12 weekly 90minute sessions EMDR (pain protocol, Grant, 1999) Taught relaxation & visualization for managing distress between sessions Pre-intervention: Short-Form Health Survey (SF-36) (higher score=better health, 0-100) State-Trait Anxiety Inventory (STAI) Beck Depression Inventory (BDI) Structured clinical interview for DSM (SCID-II) Visual Analogue Scale (VAS) Pharmacological treatment (from pain clinic) assessed as number of pills consumed SUDS (pain focused) VoC Post-intervention: Medication (number of pills consumed) SF-36, STAI, BDI, VAS	New patients to Pain clinic 55 initially 12 drop out (4 men 8 women) 38 complete (32 women 6 men) Age? Headache= 30 Fibromyalgia=4 Neuropathic pain= 4 Average pain 12 years	Wilcoxon matched-pairs signed rank test & Bonferroni correction *P*=0.00625 Pre-intervention: 6X SF-36 domains=↓50 (inc body pain) 2X SF-36 = ↑ 50 BDI= 1-30 (median 17) Trait anxiety=0-94 (median 65) General anxiety= 0-94 (median 65) VAS= 4-10 (median 8) Post-intervention: 1X SF-36 = ↓50 7X SF-36= ↓50 (inc body pain) (statistically significant improvement in all 8 domains of SF-36) BDI= 0-22 (median 9) *p*=.002 Trait anxiety=0-94 (median 51.5) *p* <.001 General anxiety= 0-94 (median 51.5) *p* <.001 VAS= 1-9 (median 6) *p*= .002 All statistically significant Medication use↓ in 79.49% (30 subjects) remainder = no change SCID-II: 73.7% sample fulfilled criteria for at least 1 axis II personality disorder (most prominent 44.7% = obsessive compulsive disorder)	Inclusion/exclusion criteria stated No long term follow-up- are results maintained? Sample bias- women & mostly Headache No control group Ages of subjects and individual duration of pain unknown- generalisability ↓ Reasons given for drop-outs CP defined using IASP classification Not know if pharmacological treatment initiated from pain clinic different/same from before pain clinic. Statistically significant pain reduction in pain Medication use↓ but diverse pharmacology manipulated by subjects thus treat with caution Use of pain EMDR protocol some detail given. Not known who delivered intervention or experience/qualifications Valid and reliable measurement tools Well written study, statistics appropriate and well-presented and suitable to answer the research question. Lacks internal and external validity. High quality study.

AUTHOR	AIM	STUDY	SUBJECTS	MAIN FINDINGS	COMMENTS
de Roos, Veenstra, Jongh, Hollander-Gijsman, Van der Wee Zitman, Van Rood, 2010	To investigate whether a psychological treatment directed at processing the emotional & somatosensory memories associated with amputation reduces PLP	Pilot study Pre-test/post-test. (no fixed number of sessions-strict ending criteria). Weekly 90min sessions. Data collected 2 weeks before intervention, post intervention, 3mth F/up, long-term F/up Clinical diagnostic interview to explore presence of axis 1 disorders. Symptom diary (2weeks) rating Pain intensity 3Xday (VAS 0-10) at 2 weeks pre-intervention, post intervention, 3 mth F/up, long-term F/up. Psychological distress- Symptom checklist (SCL-90) (scores 90-450). Fatigue-Checklist Individual Strength (CIS-20R)(20-140). PTSD symptom intensity-Impact of Events Scale (15-60) & Self-inventory List (SIL)(22-88) Health-related quality of life-short-form36 health survey (SF-36) (0-100). Standard EMDR protocol for trauma memory & pain memory. In-session PLP=pain protocol (combination of standard & Grant, 1999) Long term F/up= 26-40mths (mean 2.8yrs)	10 consecutive Participants 6 men 4 women 32-67yrs PLP following leg amputation (*n*=3, accident) (*n*=2, cancer) (*n*=2, medical failure) (*n*=1, complex regional pain syndrome (CRPS)). *n*=1, bilateral amputee *n*=4, no pre-amputation pain *n*=2 pre-amptn pain 0-6mths *n*=1 pre-amptn pain 6-12mth *n*=3 pre-amptn pain 12mth + Inclusion criteria: At least 12 mths PLP and severe disabling pain For at least 5 days A week. Exclusion criteria: If psychiatric disorder Diagnosed requiring Immediate treatment (psychosis, risk of Suicide), epilepsy, Pregnancy (due to possible complications during EMDR). Subjects asked not to Change medication Throughout study period & asked to refrain for other Treatment for PLP	Sessions completed 3-10 (mean 5.9) Data analysis=intention-to-treat basis. One-sample *t*-test detect differences between sample and general population $p \leq 0.05$ Clinical diagnostic interview: 9=normal profile 1= DSM-IV-TR criteria for PTSD, OCD, alcohol dependency, adjustment disorder & depressed mood 10=criteria for pain disorder Mean pain scores pre-test=5.0, post-test=2.8,F/up= 2.5. Pairwise comparison=significant mean pain ↓pre-post intervention (*p*=0.00) maintained at F/up 2 subjects=no improvement 4 subjects= improvement not maintained at F/up 4 subjects= improvement maintained at F/up. Those pain-free stopped medication SCL-90 & SF-36(vitality & bodily pain) = significant improvement. Other scores (except concentration subscale of CIS-20R & physical functioning of SF-36) showed improvements not maintained at F/up Long-term F/up= 6 subjects, 1 subject withdrew after 4th session, 1 died from cancer (had shown improvement), 1 damaged stump prior to F/up, 1 didn't have time to devote to data collection but stated no change from 3mth F/up. 2participants = further ↓pain intensity & pain free (0.19 & 0.71); 2 participants= pain intensity stable (1.67 & 0.20); 1 participant= ↑pain intensity (2.93, lower than pre-test); 1 participant = ↑pain intensity (co-morbid CRPS in healthy foot & hernia)	Excellent detail concerning Subjects descriptive data-location of amputation, descriptors of PLP, medication used, how subjects were recruited, ethical approval, informed consent. Standard Protocol described fully and targets explained (trauma memory, pain memory, and in-session PLP). Used 2 protocols (no details given for modified standard/pain hybrid) No detail of qualification level/level of experience for those administering EMDR Missing data explained with rationale for substitution Side effects of EMDR discussed 4 subjects lost to long-term F/up EMDR effective for some but not others-different results to other studies with across board improvements generally N.B. the 2 non-responders to treatment didn't have explicit amputation-related memory laden with affect (regarding amputation as necessary life-saving event). Non-responders also didn't have any pre-amputation pain N.B. different protocol used. Small sample, no control. Well written study, statistics appropriate, well presented and suitable to answer the research question. Shows some internal and external validity. High quality study.